A systematic review of the relationships amongst cognitive control and depressive symptoms in adolescence, and an empirical study of the relationships between motor control and cognitive control in childhood

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Preface

Struggling to control thinking can involve finding it hard to stop unhelpful lines of thought and being less able to resist distraction (i.e., cognitive inhibition), having trouble switching between different frames of mind (i.e., cognitive flexibility), and struggling to remember and use information during tasks (i.e., working memory). Difficulties with controlling thinking are linked with emotional and behavioural problems in childhood, including depression and attention deficit hyperactivity disorder (ADHD).

Depression is a particularly common emotional problem in adolescence, and it can lead to poor educational, occupational, and health outcomes across the lifespan. So far, studies have looked at how groups of adolescents with and without depression do on tasks that measure how well they control thinking. These studies have found some evidence that adolescents with a diagnosis of depression struggle with controlling thinking. However, adolescents do not fit neatly into “depressed” and “nondepressed” groups. Neither is controlling thinking (i.e., through cognitive inhibition, cognitive flexibility, and working memory) an all or nothing ability. Adolescents have varying levels of depressive symptoms and skills to control thinking. However, it is unknown how varying levels of depressive symptoms and skills to control thinking relate to each other. In Paper 1, a systematic literature review including four meta-analyses was conducted. 19 individual studies, involving over 3700 children, which looked at relationships between depressive symptoms and controlling thinking were reviewed to work out which aspects of controlling thinking are linked with depressive symptoms. The results suggested that the ability to resist distraction, and working memory, or the ability to keep and manipulate information held in the mind’s eye are linked with depressive symptoms. Therefore, therapies which improve adolescents’ skills to resist distraction and keep and manipulate information held in mind might be helpful additions to existing evidence-based therapies for depression. Although, these findings are
limited by the typically poor quality of the source studies and a lack of consideration of relevant covariates, such as levels of anxiety symptoms.

ADHD is a relatively common neurodevelopmental problem in childhood which is associated with poor cognitive, educational, and occupational outcomes. Children with ADHD experience difficulties with controlling thinking and controlling movement. Children learn to control movement before they learn to control thinking, leading some researchers to suggest that difficulties with controlling movement may result in difficulties with controlling thought in ADHD. However, the relationship between controlling movement and controlling thought is poorly understood. One reason for this is that controlling movement, like controlling thought, involves several specific skills. Specific movement control skills include being able to choose to generate movements (i.e., motor generation), being able to adjust movements in response to unchanging visual stimuli (i.e., visuomotor fluency), and being able to control movement in changeable visual situations, for example when an object to be followed moves in an unpredictable way (i.e., visuomotor flexibility). Also, skills for controlling thinking involve several more basic skills which act as building blocks (e.g., information processing efficiency, a speed-accuracy trade-off, and the time necessary for stimuli encoding and motor response preparation). In Paper 2, to work out which specific movement skills (i.e., motor generation, visuomotor fluency, and visuomotor flexibility) are linked with skills for controlling thinking (i.e., cognitive inhibition, working memory, and cognitive flexibility) in childhood, 255 children aged 4 to 10 were assessed with a range of movement and thinking tasks. Cognitive modelling was also used to break down the thinking skill of resisting distraction (cognitive inhibition) into its basic building blocks, including how long it takes children to process information (i.e., “drift rate”), whether they prioritise doing the task quickly or doing the task accurately (i.e., “boundary separation”), and the time taken to encode stimuli and prepare motor responses (i.e., “nondecision time”). Carers were
interviewed to measure the levels of hyperactive-impulsive ADHD symptoms displayed by the children. The results showed that being able to control movement in response to unchanging visual stimuli (visuomotor fluency) is linked with skills in resisting distraction (cognitive inhibition) and being able to control movement in changeable visual situations (visuomotor flexibility) is linked with skills in switching between different frames of mind (cognitive flexibility). These results were the same regardless of whether children showed high or low levels of hyperactive-impulsive ADHD symptoms. Together, these findings suggest that therapies which improve skills in controlling movement in response to visual changes might reduce distractibility and enhance the skill to switch between different frames of mind in young children, regardless of whether they show high or low levels of hyperactivity. Unfortunately, these findings are cross sectional which means they cannot demonstrate causality. Further research is needed to determine whether visuomotor fluency and visuomotor flexibility causally influence cognitive inhibition and cognitive flexibility, respectively.

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The Relationship Between Cognitive Control and Depressive Symptoms in Adolescence:
A Systematic Review and Meta-analysis

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This paper was prepared in accordance with submission guidelines for the Journal of Child and Adolescent Mental Health (see Appendix E); however, the word count for the Doctorate of Clinical Psychology (8,000 words maximum) was adhered to. **Word count**: Abstract 250; main text 6709 (excluding figures/tables).
Abstract

Background: Depressive symptoms are common in adolescence and associated with poor outcomes. Cognitive control may play an important role. Previous syntheses have adopted a categorical approach, finding some evidence for cognitive control deficits in adolescents with depression compared to those without. However, a synthesis of individual differences in depressive symptoms and cognitive control, in line with a dimensional approach, had not yet been attempted. Method: This systematic review and meta-analysis synthesised associations between objectively measured aspects of “cold” and “hot” cognitive control and depressive symptoms (total k = 18). Meta-analyses of cognitive flexibility (k = 7, N = 1131), inhibition of distraction (k = 3, N = 213), inhibition of overlearned responses (k = 4, N = 359), and working memory (k = 3, N = 783) were conducted. A systematic review of behavioural inhibition and “hot” cognitive control and depressive symptoms was performed. Results: Studies were clinically, methodologically, and statistically heterogeneous, and generally of low quality. There was evidence in favour of small associations between inhibition of distraction and depressive symptoms and auditory-verbal working memory and depressive symptoms. Also, there was mixed evidence regarding associations between depressive symptoms and “hot” cognitive control. Conclusions: The abilities to resist the effects of distracting information (cognitive inhibition of distraction) and maintain and manipulate information in the mind’s eye (working memory) are associated with depressive symptoms in adolescence. These findings are broadly consistent with theoretical models of adult depression and have implications for understanding, assessing, and treating depression and cognitive control difficulties in adolescence.

Key words: Cognitive Control; Depression; Adolescence; Dimensional; Individual Differences
Key Practitioner Message

- What is known? There is some evidence that adolescents with a diagnosis of depression display cognitive control deficits. However, it is unclear whether individual differences in depressive symptoms and cognitive control are linked across the continuum of depressive symptoms in adolescents.

- What is new? This systematic review and meta-analysis reports evidence for links between individual differences in the abilities to resist distraction (cognitive inhibition of distraction) and to maintain and manipulate auditory-verbal information in the mind’s eye (working memory) and individual differences in depressive symptoms in adolescence.

- What is significant for clinical practice? These findings suggest that adult models of depression focusing on inhibition and working memory are also relevant to adolescents, and that interventions targeting cognitive inhibition of distraction and working memory may be useful adjuncts for the treatment of depressive symptoms in adolescence.
Introduction

Depression is the most common mental health problem experienced by adolescents (Thapar et al., 2012). Prior to the outbreak of COVID-19, the global prevalence of clinically significant depressive symptoms in adolescence was approximately 12%, but this rate may have doubled during the pandemic (Racine et al., 2021). In adulthood, prevalence may be as high as 25% (Torre et al., 2021). Depressive symptoms are known to negatively impact on important educational (Fletcher, 2010) and health outcomes (Keenan-Miller et al., 2007) of adolescents. Further, half of those adolescents who completed suicide meet criteria for a diagnosis of depression (Hawton & Heeringen, 2009). Subsequently, there is an important clinical need to understand why depressive symptoms arise and how they are maintained in adolescence.

Unfortunately, despite their prevalence and the poor outcomes associated with them, depressive symptoms in adolescence are only partially understood. Some authors argue that adolescent depression is equivalent to adult depression (Bernaras et al., 2019), while others look to the unique biopsychosocial contexts which accompany adolescence to explain its frequent onset during this period (Kessler et al., 2005). At the biological level, the adolescent brain undergoes several developmental processes, including myelination, neuronal pruning, and the growth of dorsolateral and orbitofrontal prefrontal cortex, which facilitate greater cognitive and social functioning (Blakemore & Choudhury, 2006; Poletti, 2009). At the psychological level, adolescents develop greater cognitive capabilities including for abstract thinking, acknowledging the beliefs of others, and increased introspection (Remschmidt, 1994). Socially, adolescents must navigate new peer groups and social hierarchies as they transition to secondary school (Blakemore & Mills, 2013). All these factors may interact to produce risk and resilience for depressive symptoms in adolescence (Thapar et al., 2012).
One important biopsychosocial factor in understanding adolescent depression is cognitive control or executive functioning. While cognitive control is biologically based and psychologically characterised, it is also important for navigating the social world (Blakemore & Choudhury, 2006; Miyake & Friedman, 2012). Broadly speaking, cognitive control refers to effortful, top-down cognitive processes which enable adolescents to plan, direct their attention, and override overlearned or instinctual responses (Diamond, 2013). Cognitive control can be stratified into three separable but related processes (Miyake et al., 2000).

“Cognitive flexibility” involves the ability to shift attention from task to task or between lines of thought, preventing perseveration on tasks or thoughts that are no longer relevant to a goal. “Inhibition” involves overriding overlearned, habitual responses and resisting the effects of distraction. Further, it is possible to distinguish between cognitive (inhibiting thoughts) and behavioural (inhibiting actions) inhibition (Stein et al., 2017). “Updating” involves refreshing and monitoring the content of working memory or the mind’s eye. These aspects of cognitive control are sometimes referred to as “cold” cognitive control because they involve the regulation of thinking rather than emotion or affective information.

Cognitive models of depression (e.g., Abramson et al., 1978; Beck, 1987; Hankin, 2008; Horowitz et al., 2007) highlight persistent negative thoughts as a central factor in the genesis and maintenance of depressive symptoms. The persistent nature of these thoughts may be associated with difficulties regulating thinking (i.e., “cold” cognitive control). However, the relationship between specific aspects of cognitive control and depressive symptoms is poorly understood. Based on models in the adult literature, Mennies et al. (2021) draw attention to three possible relationships between “cold” cognitive control and trans-diagnostic ruminative thinking which might also apply to depressive symptoms in adolescence. First, persistent negative thinking might arise because of failure to switch one’s focus from negative self-referential information neutral to positive self-referential
information (Koster et al., 2011). This model implies that cognitive flexibility is related to depressive symptoms in adolescence. Second, rumination might arise because of difficulties with inhibiting or stopping negative information from entering the mind and not being able to expel this information (Joormann, 2010). This model suggests that inhibition and working memory are both linked with depressive symptoms (Mennies et al., 2021). Finally, persistent negative thoughts might become “stuck” in working memory because of low mood narrowing the scope of attention and preventing updating (Whitmer & Gotlib, 2013). This model indicates an association between working memory and depressive symptoms in adolescence.

The above cognitive flexibility (Koster et al., 2011), inhibition-working memory (Joorman, 2010), and working memory-based (Whitmer & Gotlib, 2013) accounts of depression are not necessarily mutually exclusive. This is because there appears to be unity as well as diversity of cognitive control abilities at the cognitive and neural levels (Cragg & Chevalier, 2012; Diamond, 2013; Miyake et al., 2000). The overlap amongst different aspects of cognitive control suggests that a relationship between domain general cognitive control and depressive symptoms is possible. Alternatively, more specific relationships between depressive symptoms and aspects of cognitive control might be present. Regarding inhibition, for example, it is possible to distinguish between inhibiting interference from distracting stimuli, inhibiting interference from habitual or overlearned responses, and inhibiting motor actions (Kornblum, 1994; Paap et al., 2020). Depressive symptoms may be associated with a specific aspect of inhibition in adolescence.

In contrast with “cold” cognitive control, “hot” cognitive control involves skills such as managing behaviour in the context of reward, delaying gratification, and regulating emotions (Poon, 2018; Zelazo et al., 2010). Reviews associate “hot” cognitive control with orbitofrontal cortex and “cold” cognitive control with dorsolateral prefrontal cortex.
(Salehinejad et al., 2021; Zelazo & Carlson, 2012). One neuropsychological theory of depression based on neuroimaging research suggests that adolescent depression is more related to orbitofrontal but not dorsolateral mediated aspects of cognitive control (Poletti, 2009). Difficulties with “hot” aspects of cognitive control such as regulating emotions and making reward-guided decisions may relate to adolescents’ difficulties with inhibiting sad mood, emotional decision making, and withdrawal from positively reinforcing activities (Berle & Moulds, 2013). Unfortunately, little attention has been devoted to the study of “hot” cognitive control in adolescent depression.

To date, almost all systematic reviews and meta-analyses have used a categorical approach to cognitive control and depression. The dominant categorical approach involves synthesising performance on cognitive control tasks across groups of depressed and non-depressed adolescents. For example, Baune et al. (2014) reviewed cognitive control abilities in depressed and non-depressed young people aged 12-25 years of age and found evidence of cognitive control deficits in the clinical group. Vilgis et al. (2015) also made a categorical distinction between clinical and non-clinical groups of the same age but found that cognitive control abilities were largely intact in both groups. Neither of these studies performed meta-analytic synthesis. Goodall et al. (2018) and Wagner et al. (2015a) conducted meta-analyses of cognitive control abilities across groups of depressed and non-depressed adolescents, which together suggest deficits in inhibition, planning, and attention in clinical groups. One relevant prior systematic review did employ a continuous approach to repetitive negative thinking regarding rumination, worry, obsessions, and post-event processing (Mennies et al., 2021). This review found that objectively measured cognitive control was not related with repetitive negative thinking; however, this absence of association might reflect over-inclusivity of constructs spanning multiple presentations including depression, obsessive-compulsive disorder, and generalised anxiety disorder.
The near ubiquitous categorical approach to adolescent depression (i.e., comparing depressed versus non-depressed groups) in existing reviews can highlight cognitive deficits, or the absence of these, in adolescents with depression. However, by focusing on diagnostic group status, the categorical approach does not explicitly account for individual differences in cognitive control and depressive symptoms. It is important not to neglect individual differences in cognitive control and depression for two reasons. First, adolescent depression could be more appropriately viewed as a continuum rather a discrete category (Hankin et al., 2015). By extension, dividing adolescents into clinical and non-clinical groups may be misleading. Similarly, there is not a clear distinction between cognitive impairment and cognitive normality. In practice, “impairment” can be operationalised in several different ways (e.g., a statistically significant group difference or a mean group score -2.0 SD below that of the control group), and very low scores amongst a few members of one group could bias the mean even if most of the group members do not struggle with cognitive control. Accordingly, stratifying adolescents into depressed or non-depressed groups and considering group differences on cognitive control tasks as evidence of impairment is problematic.

Second, it is problematic to simply assume that depression causes difficulties in cognitive control or vice versa. Cognitive control abilities and depressive symptoms may be mutually reinforcing, which the developmental cascade approach to psychopathology (Masten & Cicchetti, 2010; Morea & Calvete, 2021) and a neurocognitive model of depression in adulthood (Ahern et al., 2019) suggest is possible. The development cascade perspective proposes that developmental outcomes, such as mental health status and cognitive functioning, can be viewed as the cumulative result of many interactions amongst different levels of various systems (e.g., cognitive, emotional) over time (Masten & Cicchetti, 2010); effects amongst cognitive control and depressive symptoms could be direct or indirect and unidirectional or bidirectional. Similarly, the “hot and cold” model of
depression (Ahern et al., 2019) posits bidirectional relationships between cognitive control and depressive symptoms. “Cold” and “hot” cognitive control deficits are theorised to mutually reinforce a negative memory bias and lead to depressive symptoms, which reinforce biased memory and further diminish “cold” and “hot” cognitive resources, creating a vicious cycle. Adopting a continuum as opposed to a categorical approach avoids the presumption that depression results in cognitive control deficits or vice versa. Correlations can represent multiple underlying relationships (including causation). While adopting a continuum approach might make it harder to draw causal inferences, it is consistent with contemporary models of depression and neuropsychological functioning described above.

This systematic review and meta-analysis aimed to synthesise relationships between cognitive control and depressive symptoms to better understand which elements of cognitive control, if any, are related to depressive symptoms in adolescence. These aspects of cognitive control were inhibition (regarding distraction, overlearned responses, and motor actions), cognitive flexibility, working memory, and “hot” cognitive control. Most source studies in this review were not included in relevant previous syntheses (i.e., Baune et al., 2014; Goodall et al., 2018; Vilgis et al., 2015; Wagner et al., 2015a) using categorical or continuum (i.e., Mennies et al., 2021) approaches. In the present systematic review and meta-analysis correlations were examined rather than group differences to characterise individual differences in cognitive control and depressive symptoms, rather than deficits and disorder, in line with a dimensional approach to psychopathology (Cuthbert, 2014).
**Methods**

The systematic review was registered on PROSPERO on the 14th January 2022 (see Appendix A).

**Search terms**

The following search terms were entered into the PsycInfo, Web of Science, and Scopus databases on the 14th of January 2022 to search the abstracts, titles, and key words of records from any year: (((executive function*) OR (cognitive control)) AND ((internali*) OR (anxi*) OR (depress*)) AND (adolesc* OR teen*)). The search terms included anxiety and internalising as well as depression because they are often grouped together as “internalising” problems in childhood.

**Inclusion/exclusion criteria**

Final inclusion criteria were an objective measure of at least one aspect of cognitive control, a measure of depressive symptoms, concurrent assessment of cognitive control and depressive symptoms, bivariate correlations, or single linear regression models from which biuvariate correlations could be derived, and a mean sample age between 11 and 18 years of age. Exclusion criteria were the possibility of organic cognitive control impairment (e.g., through head injury, treatment for cancer, paediatric diabetes) or a diagnosis of learning disability. Adolescents with diagnoses of depression or other internalising problems were eligible for inclusion. However, studies making a categorical distinction between depressed and non-depressed adolescents were only eligible for inclusion providing they reported correlations between cognitive control abilities and depressive symptoms. This was to preserve the dimensional focus on individual differences in cognitive control and depressive symptoms. Studies using composite measures of cognitive control (e.g., Trail Making Test
Part B Completion Time plus Wisconsin Card Sorting Task Perseverative Errors, in Holler and Kavanaugh, 2013) were excluded to ensure purer measures of the aspects of cognitive control of interest (Lezak et al., 2012, p. 159).

**Screening**

The screening process is visualised in Figure 1 according to the most recent PRISMA guidelines (Page et al., 2021). All studies were selected by the main author. A blinded independent reviewer (a postgraduate doctoral student) also examined a subset of approximately 10% (k = 22) studies containing both included and excluded studies against the inclusion/exclusion criteria. Cohen’s kappa was 1.0 (i.e., 100% agreement was observed at full text screening for inclusion/exclusion.)

**Quality Appraisal**

The Quality Assessment Tool for Studies with Diverse Designs (QATSDD) quality appraisal tool (Sirriyeh et al., 2012) was used to appraise overall study quality (see Appendix B). The QATSDD measures sixteen criteria which are rated on a scale from zero (worst) to three (best) and summed to a total quality score. The criteria cover theoretical (e.g., explicitly theoretical background), methodological (e.g., sample size justification), and interpretative (e.g., discussion of strengths and limitations) quality. The QATSDD, which is designed for appraising diverse studies, was selected because of the heterogeneity of study designs, which included both cross sectional and cohort studies. Additionally, the QATSDD has good content validity and interrater reliability (Cohen’s kappa = 0.72; Sirriyeh et al., 2012). The lead author appraised all the included studies. A random selection of approximately 25% of the included studies (k = 5) were also rated by the same blinded independent rater who assisted with screening. Interrater reliability, calculated as an intraclass correlation
coefficient, was 0.76 (Appendix C). Disagreements were resolved by using the average of the discrepant scores.

**Data Extraction**

Study names and dates, sample sizes, cognitive control measures, depressive symptoms measures, and correlation coefficients were extracted. When youth-reported and parent-reported depressive symptoms were present (e.g., Han et al. 2016), correlations between youth-reported depressive symptoms and cognitive control were selected based on the assumption that youth-reported measures better captured subjective experiences of depressive symptoms. Where necessary, correlation coefficients were reversed so that higher scores on all cognitive control tasks reflected poorer performance in order to make meta-analysis results easier to interpret.

**Systematic Review and Meta-Analytic Synthesis Approach**

Studies were grouped by into broad cognitive flexibility, inhibition, working memory, and “hot” cognitive control categories. Within these four broad areas, subsets of suitable studies were subject to meta-analysis. Four meta-analyses were performed to synthesise correlations between depressive symptoms and 1) cognitive flexibility, 2) inhibition of distracting information, 3) inhibition of overlearned responses, and 4) working memory. Meta-analyses of studies in the behavioural inhibition and “hot” cognitive control domains was not attempted given limited number of studies assessing the same sub-construct and the variety of tasks used. For suitable collections of studies, the R-based online application Meta-Mar (https://www.meta-mar.com) was used to perform random-effects meta-analyses of correlation coefficients (Beheshti et al., 2020). A random effects model was deemed most appropriate given the heterogeneity of the samples and measures used in the field. Studies
were weighted by sample size and not quality. In the meta-analyses, neuropsychological tasks were grouped based on analogous tasks where possible (e.g., the Wisconsin Card Sorting Task and the Intra-Extra Dimensional Set Shift Task, Stroop Test and Color-Word Interference Test, etc.) or else based on the cognitive control construct they most closely probed, as is common practice in meta-analyses of neuropsychological variables (Demakis, 2006). An alpha threshold of 0.05 was set regarding statistical significance. Heterogeneity was quantified with the $I^2$ statistic; $I^2$ confidence intervals were reported as well as point estimates to mitigate potential $I^2$ bias in small meta-analyses (von Hippel, 2015). Extracted data and Meta-Mar Output is displayed in Appendix D.
Figure 1. PRISMA Flowchart of Studies Included in the Systematic Review and Meta-Analyses.

Identification of studies via databases and registers
Records identified from:
- PsycInfo (n = 1622)
- Scopus (n = 1970)
- Web of Science (n = 1096)
Records removed before screening:
- Duplicate records (n = 1426)
Records screened (n = 3262)
Records excluded (n = 3037)
Reports sought for retrieval (n = 225)
Reports not retrieved (n = 11)
Reports assessed for eligibility (n = 214)
Reports excluded (n = 198)
  - No correlations (n = 52)
  - Depression treated as categorical (n = 37)
  - Mean age < 11 years (n = 21)
  - Subjective executive functioning measures (n = 16)
  - Mean age > 18 years (n = 15)
  - No measure of depression (n = 15)
  - Not peer reviewed (n = 15)
  - Not empirical (n = 12)
  - No concurrent assessment of depression and executive functioning (n = 6)
  - Potential organic executive dysfunction (n = 4)
  - Duplicate dataset (n = 3)
  - Composite executive functioning score (n = 2)
Studies included in review (n = 19)

Identification of studies via other methods
Records identified from:
- Personal database (n = 3)
Reports sought for retrieval (n = 3)
Reports not retrieved (n = 0)
Reports assessed for eligibility (n = 3)
Reports excluded (n = 0)
Results

The included studies (k = 18) are summarised in Table 1. The median sample size was 149 (MAD = 79). The mean age of adolescents across studies was 14.26 (SD = 1.58). On average, 51.8% of adolescents were female. All studies were conducted in the United States, Europe, or Australia and most adolescents were Caucasian (68.0%). Socioeconomic status data was missing for most studies, but the available data suggested that adolescents were generally from middle class backgrounds. Eight studies included adolescents with a diagnosis of depression (Dickson et al., 2016; Han et al., 2012; Jandrić et al., 2021; Kavannaugh et al., 2012; Peters et al., 2019; Sommerfeldt et al., 2015; Valentino et al., 2012; Wagner et al., 2015b).

Cognitive flexibility and depressive symptoms

Ten studies assessed cognitive flexibility (Dickson et al., 2017; Evans et al., 2015; Han et al., 2016; Morea & Calvete, 2021; Murphy et al., 2018; Rifkin et al., 2021; Stewart et al., 2018; Valentino et al., 2012; Vergara-Lopez et al., 2013; Wagner et al., 2015b). Average study quality was relatively low (64.1% on the QATSDD), particularly concerning justification for sample size and adequate assessment of the reliability of measures. There was also evidence of considerable clinical (see Table 1) and methodological heterogeneity. The studies used a diverse array of tasks including the Wisconsin Card Sorting Task (WCST), the Intra-Extra Dimensional Set Shift Task (IED), The Creature Counting subtest from the Test of Everyday Attention for Children, the Changes Cognitive Flexibility Task, and the Internal Switch Task. The WCST and IED were the most employed tasks, being used in seven of the ten relevant studies. Given their similarity as card sorting tasks generating perseverative error scores (i.e., failures to shift attentional set from irrelevant material), these tasks were entered into a meta-analysis shown in Figure 2.
<table>
<thead>
<tr>
<th>Study</th>
<th>Mean age (SD) and range</th>
<th>Female (%)</th>
<th>Design</th>
<th>Sample size and study groups</th>
<th>Country and ethnicity</th>
<th>Socio-economic status (SES)</th>
<th>Cognitive control constructs and measure(s)</th>
<th>Depressive symptoms measure(s)</th>
<th>Study quality (QATSDD Checklist)</th>
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</table>
| Davidovich et al. (2016) | 13.77 (2.04) 8 - 18     | 59.7%      | Cross sectional | 187 with a parent with recurrent depression | Wales, United Kingdom; ethnicities unknown | Unknown                      | “Hot” cognitive control
- Affective GNG                   | CAPA Depression, interview, parent and adolescent completed or parent-completed only | 53.6% |
| Dickson et al. (2017)   | 17.77 (0.46) 16 - 18     | 64%        | Cross sectional | 86 general population (14% self-reported mental health diagnosis) | United States - 84.9% Caucasian
- 5.8% African American
- 4.7% Mixed race
- 2.4% Hispanic
- 2.3% Other | Unknown                      | Cognitive flexibility
- WCST                  | CES-D, self-reported                | 59.5% |
| Evans et al. (2016)     | 12.36 (1.77) 9 - 15     | 52.1%      | Cross sectional | 192 general population | United States - 71.4% Caucasian
- 18.2% African American
- 2.6% Asian-American
- 3.6% Hispanic
- 4.2% Mixed race | Unknown                      | Cognitive flexibility
- WCST                  | CDI, self-reported                | 59.5% |
| Gray et al. (2016)      | Maltreated: 13.51 (1.75) 12 – 15
Non-maltreated: 13.90 (1.50) | 54%        | Cross sectional | 51 adolescents in care:
- 24 un-maltreated
- 27 maltreated | Australia; ethnicities unknown | Unknown                      | Inhibition (distraction)
- Bespoke non-emotional attentional control task | CDI, self-reported                | 65.5% |
<table>
<thead>
<tr>
<th>Study</th>
<th>Mean (SD)</th>
<th>Age Range</th>
<th>Methodology</th>
<th>Sample Characteristics</th>
<th>Measure/Control</th>
<th>Cognitive Flexibility</th>
<th>SES</th>
<th>Inhibition/Effort</th>
<th>Control</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Han et al.</td>
<td>13.67 (1.52)</td>
<td>12 - 15</td>
<td>Cohort</td>
<td>220 general population with internalising symptoms overrepresented United states - 70% Caucasian - 30% Non-Caucasian</td>
<td>Most</td>
<td>DISC-IV, mother- and self-reported</td>
<td>Sodium</td>
<td>WCST</td>
<td>45.3%</td>
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<tr>
<td>(2016)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>middle-to-upper middle SES</td>
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<tr>
<td>Han et al.</td>
<td>17.39 (1.58)</td>
<td>16 - 19</td>
<td>Cross sectional</td>
<td>61 general population: - 30 no depression - 31 depression United States - 73.3% Caucasian - 26.7% Black and Minority Ethnic</td>
<td>Unknown</td>
<td>BDI-II, self-reported</td>
<td>52.4%</td>
<td>(distraction)</td>
<td>- ANT</td>
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<td>(2012)</td>
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<td>“Hot” cognitive control</td>
<td>- IGT</td>
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<td></td>
<td></td>
<td></td>
<td>- face GNG</td>
<td></td>
</tr>
<tr>
<td>Jandrić et al.</td>
<td>15.09 (1.6)</td>
<td>13 - 17</td>
<td>Cross sectional</td>
<td>100 referred for diagnostic assessment: - 59 neurotic, stress-related and somatoform disorders - 38 emotional disorders with onset specific to childhood - 3 depressive episode Croatia; ethnicities unknown</td>
<td>Unknown</td>
<td>BDI-II, self-reported</td>
<td>57.1%</td>
<td></td>
<td>- CANTAB IED</td>
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<td>(2021)</td>
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<tr>
<td>Study</td>
<td>Mean (SD)</td>
<td>Age Range</td>
<td>Prevalence</td>
<td>Study Design</td>
<td>Population Description</td>
<td>Country</td>
<td>Ethnicity</td>
<td>Cognitive Flexibility</td>
<td>Depression Measure</td>
<td>CDI, Self-reported</td>
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<tr>
<td>Kavanaugh et al. (2012)</td>
<td>15.33 (1.3)</td>
<td>13–18</td>
<td>43%</td>
<td>Cross-sectional</td>
<td>105 with mood disorders: - 61 without depression - 41 without depression</td>
<td>United States; ethnicities unknown</td>
<td>Unknown</td>
<td>Cognitive flexibility - WCST</td>
<td>Inhibition (automatic responses) - Stroop</td>
<td>CDI, self-reported</td>
</tr>
<tr>
<td>Kim et al. (2021)</td>
<td>12.73 (2.57)</td>
<td>8–16</td>
<td>46.4%</td>
<td>Cohort</td>
<td>237 general population with around 50% exposed to maltreatment</td>
<td>United States; ethnicities unknown</td>
<td>Mean income-to-needs ratio 3.33 (SD = 2.73), above poverty line of 1.0.</td>
<td>Inhibition (automatic responses) - DNA-II Arrows</td>
<td>CDI, self-reported</td>
<td>67.9%</td>
</tr>
<tr>
<td>Morea &amp; Calvete (2021)</td>
<td>14.59 (1.36)</td>
<td>12-17</td>
<td>41%</td>
<td>Cohort</td>
<td>698 general population</td>
<td>Spain; ethnicities unknown</td>
<td>- 12.5% low - 12.3% low-medium - 26.4% medium - 21.5% high-medium - 18.8% high - 8.6% no data</td>
<td>Cognitive flexibility - CCFT</td>
<td>CES-D, self-reported</td>
<td>70.2%</td>
</tr>
<tr>
<td>Moreno-Manso et al. (2020)</td>
<td>14.98 (1.0)</td>
<td>8-12</td>
<td>47.5%</td>
<td>Cross-sectional</td>
<td>61 in residential care: - 33 were victims of abuse - 28 no history of abuse</td>
<td>Spain; ethnicities unknown</td>
<td>Unknown</td>
<td>Inhibition (automatic responses) - Color and Word Test (Stroop)</td>
<td>SENA Depression, self-reported</td>
<td>71.4%</td>
</tr>
<tr>
<td>Study</td>
<td>Mean (SD)</td>
<td>Age Range</td>
<td>Sample Description</td>
<td>Country</td>
<td>Ethnicity Distribution</td>
<td>Household Income</td>
<td>Outcome Measure(s)</td>
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<tr>
<td>Murphy et al. (2018)</td>
<td>11.94 (2.49)</td>
<td>9 – 17</td>
<td>106 general population; excluded those with clinical symptoms</td>
<td>United States</td>
<td>88.76% Caucasian, 6.52% African American, 4.68% Other</td>
<td>&lt;$60,001</td>
<td>Cognitive flexibility - CANTAB IED, Inhibition (motor) - SST</td>
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<tr>
<td>Peters et al. (2019)</td>
<td>14.58 (1.52)</td>
<td>12 - 17</td>
<td>70 from outpatient psychiatry clinics: 30 healthy controls, 18 met criteria for depression, 22 met criteria for depression and childhood trauma</td>
<td>United States</td>
<td>59.0% Caucasian, 20.3% African American, 11.9% Asian, 1.2% Native American, 7.6% Other</td>
<td>&lt;$60,000</td>
<td>Inhibition (motor) - Parametric GNG, Working memory - Spatial span task</td>
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<tr>
<td>Rifkin et al. (2021)</td>
<td>13.15 (0.99)</td>
<td>12 - 14</td>
<td>364 general population</td>
<td>United States</td>
<td>40.1% Caucasian, 56% African American, 3.9% Hispanic</td>
<td>Qualified for free school meals</td>
<td>Cognitive flexibility - TEA-Ch CC, CDI, self-reported</td>
<td></td>
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<tr>
<td>Study</td>
<td>Mean (SD)</td>
<td>Age Range</td>
<td>Sample Size</td>
<td>Sample Description</td>
<td>Location</td>
<td>Ethnicity</td>
<td>Cognitive Flexibility</td>
<td>Assessment</td>
<td>Depression</td>
<td>Other</td>
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<tr>
<td>Sommerfeldt et al. (2015)</td>
<td>16.6 (1.9)</td>
<td>12 - 20</td>
<td>162</td>
<td>Cross sectional</td>
<td>United States</td>
<td>- 72.2% Asian</td>
<td>Inhibition (distraction)</td>
<td>BDI-II, self-reported</td>
<td></td>
<td>68.5%</td>
</tr>
<tr>
<td>Stewart et al. (2018)</td>
<td>13.85 (0.78)</td>
<td>13 - 16</td>
<td>149</td>
<td>Cohort</td>
<td>Scotland, United Kingdom; ethnicities unknown</td>
<td>16% free school meals</td>
<td>Cognitive flexibility</td>
<td>BDI-II, self-reported</td>
<td></td>
<td>64%</td>
</tr>
<tr>
<td>Valentino et al. (2012)</td>
<td>14.1 (2.3)</td>
<td>7 – 17</td>
<td>49</td>
<td>Cross sectional</td>
<td>United States</td>
<td>- 43.6% Caucasian</td>
<td>Inhibition (automatic responses)</td>
<td>CDI, self-reported</td>
<td></td>
<td>32.7%</td>
</tr>
<tr>
<td>Vergara-Lopez et al. (2013)</td>
<td>13.13 (0.61)</td>
<td>13 - 14</td>
<td>373</td>
<td>Cohort</td>
<td>United States</td>
<td>- 82.6% Caucasian</td>
<td>Inhibition (motor)</td>
<td>YSR Depression, self-reported</td>
<td></td>
<td>55%</td>
</tr>
<tr>
<td>Wagner et al. (2015b)</td>
<td>12.88 (0.62)</td>
<td>12 - 13</td>
<td>486</td>
<td>Cohort</td>
<td>United States</td>
<td>- 48.8% Caucasian</td>
<td>Cognitive flexibility</td>
<td>CDI, self-reported</td>
<td></td>
<td>52.7%</td>
</tr>
<tr>
<td>depressive disorder</td>
<td>- 47.1% African American</td>
<td>- 4.2% Other</td>
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<td></td>
<td>- WISC-IV Digit Span</td>
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</tbody>
</table>

This random-effects meta-analysis (k = 7, N = 1131) did not find a statistically significant association between perseverative errors and depressive symptoms in adolescents (r = 0.14, 95% CI = 0.00, 0.28, p = 0.053). Quantifiable heterogeneity was high (I² = 76.4%, 95% CI: 50.5%, 88.8%). Only the largest two of the synthesised correlation coefficients were statistically significant in the source studies (Dickson & Ciesla, 2018; Valentino et al., 2012). Non-included studies utilising Creature Counting (Rifkin et al., 2021; Wagner et al., 2015b), the Changes Cognitive Flexibility Task (Morea & Calvete, 2021), and the Internal Switch Task (Stewart et al., 2011) all reported non-significant findings.

Figure 2. Meta-analysis of Correlations between Perseverative Errors (Cognitive Flexibility) and Depressive Symptoms.

<table>
<thead>
<tr>
<th>Study</th>
<th>Weight (common)</th>
<th>Weight (random)</th>
<th>Correlation IV, Fixed + Random, 95% CI</th>
<th>Correlation IV, Fixed + Random, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dickson et al. (2017)</td>
<td>186</td>
<td>7.5%</td>
<td>13.2%</td>
<td>0.48 [0.30; 0.63]</td>
</tr>
<tr>
<td>Evans et al. (2015)</td>
<td>192</td>
<td>17.0%</td>
<td>15.6%</td>
<td>0.12 [-0.02; 0.26]</td>
</tr>
<tr>
<td>Han et al. (2016)</td>
<td>220</td>
<td>19.5%</td>
<td>15.9%</td>
<td>0.06 [-0.07; 0.19]</td>
</tr>
<tr>
<td>Kavanauagh et al. (2012)</td>
<td>105</td>
<td>9.2%</td>
<td>13.9%</td>
<td>-0.05 [-0.24; 0.14]</td>
</tr>
<tr>
<td>Murphy et al. (2018)</td>
<td>106</td>
<td>9.3%</td>
<td>13.9%</td>
<td>0.09 [-0.10; 0.28]</td>
</tr>
<tr>
<td>Valentino et al. (2012)</td>
<td>49</td>
<td>4.1%</td>
<td>10.7%</td>
<td>0.38 [0.11; 0.60]</td>
</tr>
<tr>
<td>Vergara-Lopez et al. (2013)</td>
<td>373</td>
<td>33.3%</td>
<td>16.8%</td>
<td>0.00 [-0.10; 0.10]</td>
</tr>
<tr>
<td>Total (fixed effect, 95% CI)</td>
<td>1131</td>
<td>100.0%</td>
<td>--</td>
<td>0.09 [0.03; 0.15]</td>
</tr>
<tr>
<td>Total (random effects, 95% CI)</td>
<td>--</td>
<td>100.0%</td>
<td>0.14 [0.00; 0.28]</td>
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</tbody>
</table>

Heterogeneity: Tau² = 0.0309; Chi² = 25.44, df = 6 (p < 0.01); I² = 76%
Inhibition and depressive symptoms

Seven studies assessed an aspect of inhibition (Gray et al., 2016; Kavanaugh & Holler, 2012; Kim et al., 2021; Murphy et al., 2018; Peters et al., 2019; Sommerfeldt et al., 2015; Valentino et al., 2012). Overall, studies measuring inhibition were of low quality (average QATSDD rating = 60.7%), especially regarding sample size justification. There was also evidence of clinical (see Table 1) and methodological heterogeneity, especially regarding measures. The studies used diverse tasks which, broadly speaking, probed cognitive inhibition of overlearned or habitual responses (e.g., Stroop Test, Color-Word Interference Test, Color and Word Test, and the Arrows Task), cognitive inhibition of distraction (e.g., Attention Network Test and a bespoke non-emotional attentional control task), and behavioural inhibition (Stop Signal Tasks, Go NoGo Task). Two random-effects meta-analyses were conducted to synthesise evidence for relationships between the two aspects of cognitive inhibition given similar task demands.

Figure 3. Meta-analysis of Correlations between Inhibition of Overlearned Responses and Depressive Symptoms.
The first random-effects meta-analysis (k = 4; N = 359) shown in Figure 3 suggests that inhibition of interference from overlearned or habitual responses is not related to depressive symptoms in adolescence (r = 0.08, 95% CI = -0.02, 0.19, p = 0.11). Quantifiable heterogeneity was potentially high (I^2 = 0.0%, 95% CI = 0.0%, 84.7%), noting that I^2 point estimates are biased in small meta-analyses (von Hippel, 2015). In contrast, the second random-effects meta-analysis (k = 3; N = 213) in Figure 4 suggests that inhibition of interference from distracting stimuli (p = 0.001) is small (Cohen, 1988), but significantly, related with depressive symptoms (r = 0.21, 95% CI = 0.08, 0.34). Quantifiable heterogeneity was potentially high (I^2 = 0.0%, 95% CI = 0.0%, 89.6%).

Figure 4. Meta-analysis of Correlations Between Inhibition of Distraction and Depressive Symptoms.

Note: Gray et al. (2016) featured separate groups of adolescents exposed to maltreatment or not exposed to maltreatment; therefore, the inclusion of correlations between inhibition of distraction and depressive symptoms in each group in this meta-analysis does not violate the assumption of independent effect sizes (Harer et al., 2021).
Tasks measuring behavioural inhibition were too few and heterogeneous to subject to meta-analysis. Murphy et al. (2018) used a Stop Signal Task (SST) which requires an adolescent to stop a response they have already initiated, and Peters et al. (2019) used a Go NoGo (GNG) Task which requires that they withhold a response. GNG tasks and SST rely on different neurocognitive mechanisms (Littman & Takács, 2017; Raud et al., 2020). Both studies reported no statistically significant associations with depressive symptoms (GNG – CDRS, $r = -0.07$, $p > 0.05$; SST – CBCL Withdrawn/Depression, $r = -0.06$, $p = 0.52$). Overall individual study quality was poor (QATSDD = 64.3% and 59.5%).

**Working memory and depressive symptoms**

Four studies measured working memory (Evans et al., 2015; Kavanaugh et al., 2012; Murphy et al., 2018; Wagner et al., 2015b). Studies were generally of poor quality, averaging 64.2% on the QATSDD. The main limitations were insufficient justification for sample size and insufficient assessment of measurement reliability. Clinical (see Table 1) and methodological heterogeneity was also evident. The tasks used to measure working memory were diverse, being Digit Span, Sentence Reading, and Spatial Span, but they were divisible into auditory-verbal and visuo-spatial domains. Correlation coefficients for the relationship between depressive symptoms and working memory tasks in the auditory-verbal domain were synthesised in a meta-analysis ($k = 3$, $N = 783$), shown in Figure 5. The random-effects meta-analysis was statistically significant ($r = 0.08$, 95% CI = 0.01, 0.15, $p = 0.01$). Quantifiable heterogeneity was potentially high ($I^2 = 0.0\%$, 95% CI = 0.0%, 89.6%). A single study used a working-memory task in the visuo-spatial domain (Murphy et al., 2018), finding no association between visuo-spatial working memory and depressive symptoms in adolescence ($r = -0.02$, $p = 0.87$).
“Hot” cognitive control and depressive symptoms

Five studies assessed “hot” cognitive control (Davidovich et al., 2016; Gray et al., 2016; Han et al., 2012; Kim et al., 2021; Stewart et al., 2018). Mean overall study quality for studies assessing “hot” cognitive control was relatively poor (QATSDD = 62.87%). Studies were particularly limited regarding sample size justification and adequate consideration of the reliability of measures. The measures demonstrate high methodological heterogeneity and participant characteristics in Table 1 suggest considerable clinical heterogeneity. Given the diversity of tasks used and functions probed, meta-analysis was not appropriate and so a narrative review was conducted.

Both Davidovich et al. (2016) and Han et al. (2012) measured “hot” behavioural inhibition with emotional GNG tasks. Davidovich et al. (2016) presented adolescents with rapidly changing positive or negative words (e.g., “happy” or “sad”) and instructed them to respond only to positive or negative words and not to respond to words of the other valence.
Performance on this task was not significantly correlated with depressive symptoms (r = .06, p > 0.05). In a sample of adolescents with a diagnosis of depression, Han et al. (2012) used a facial GNG task in which adolescents had to respond to “go” emotional expressions (fearful, happy, or calm) and inhibit responses to “no-go” expressions (fearful, happy, or calm). They found several moderately sized associations between performance on anger versus neutral, fear versus neutral, and sadness versus neutral trials on a facial GNG task and depressive symptoms (r’s = 0.421 – 0.487, p’s < 0.05). In this group with a diagnosis of depression, adolescents with higher depressive symptoms reacted more quickly in response to emotional face stimuli than to neutral face stimuli. The authors interpreted this finding as evidence of an attentional bias towards negative stimuli in adolescent depression, rather than an issue with behavioural inhibition in the context of emotion per se.

Han et al. (2012) also measured risky decision making with the Iowa Gambling Task in which adolescents had to rely on implicit somatic markers to select low-reward, low-risk options leading to net financial gain over between high-reward, high-reward options which led to net loss. No significant associations were present between the Iowa Gambling Task and depressive symptoms in adolescents with a diagnosis of depression. Kim et al. (2021) measured “hot” inhibition in a non-clinical sample, half of which had been exposed to maltreatment. They used an emotional Stroop task in which adolescents were presented with an image of a happy or fearful face with the word “happy” or “fear” overlaid. In congruent (incongruent) trials, the word matched (did not match) the facial expression. They did not find a significant association between the inhibition of overlearned responses in the context of emotion and depressive symptoms (r = -0.03, p > 0.05).

Gray et al. (2016) used measured attentional control following exposure to distressing emotions with a bespoke task in which adolescents were presented with a threatening (angry or fearful) face and timed while identifying a target letter (e.g., X) in two strings of letters
There was evidence of an association between attentional control in the context of emotional faces and depressive symptoms measured by the CDI in adolescents who had experienced maltreatment ($r = -0.358$, $p = 0.012$), although this association was no longer significant when data from the entire sample of maltreated and un-maltreated adolescents were analysed (Gray et al., 2016). The authors interpreted this association as evidence of an attentional bias away from angry facial expressions in adolescents with depression.

Stewart et al. (2018) measured “hot” cognitive flexibility by presenting adolescents with male and female faces showing angry or neutral expressions, instructing them to keep track of the number of faces depending on a condition (e.g., the number of angry faces), before switching this condition (e.g., from counting the number of angry faces to the number of neutral faces) and then calculating reaction time differences between these. They found a small but statistically significant association between “hot” cognitive flexibility, measured by reaction time cost while switching focus between angry and neutral faces and depressive symptoms ($r = -0.14$, $p < 0.05$). That is, adolescents who were switched their focus between angry and neutral faces faster displayed more depressive symptoms. The study was of relatively high overall quality (QATSDD = 76.2%) in comparison with the other studies. However, the association between “hot” cognitive flexibility and depressive symptoms was no longer significant at 6-month follow-up.
Discussion

This systematic review and meta-analysis synthesised relationships between cognitive control (cognitive flexibility, cognitive inhibition, working memory, and “hot” cognitive control) and depressive symptoms to investigate which elements of cognitive control, if any, are related to depressive symptoms in adolescence. The meta-analysis adopted a dimensional, as opposed to a categorical, approach to depression. A meta-analysis found support for a small association between the “cold” inhibition of distracting information and depressive symptoms in adolescence. Adolescents with better inhibition of distraction displayed fewer depressive symptoms. This finding is broadly consistent with the inhibition-working memory account of depressive symptoms which states that deficits in cognitive inhibition lead to difficulty stopping negative material from entering working memory and difficulty with expelling negative material from working memory (Joormann, 2010). Although, it is based on a small number of correlations and hampered by poor study quality and high clinical heterogeneity. Indeed, only one source correlation was statistically significant (Sommerfeldt et al., 2015). In contrast, meta-analytic evidence was not found in favour of an association between inhibition of overlearned responses and depressive symptoms. Additionally, there was no systematic review evidence for a link between behavioural inhibition and depressive symptoms; although, this section of the review was also undermined by the small number of source studies available for consideration. Together, these results suggest specific difficulties with inhibition in the context of distraction are smally associated with depressive symptoms in adolescence. For example, adolescents with higher levels of depressive symptoms may struggle not to find negative automatic thoughts distracting. Subsequently, the inhibition account of depressive symptoms (Joorman, 2010), which focuses on general cognitive inhibition, could be adapted to focus on this specific form of “cold” cognitive inhibition.
A further meta-analysis suggested that there is a significant association between auditory-verbal working memory and depressive symptoms in adolescence. This finding is consistent with the notion that auditory-verbal working memory is linked with depressive symptoms in adolescence, as predicted by the working-memory account of persistent negative thinking (Whitmer & Gotlib, 2013) and, to a lesser extent, the inhibition-working memory account of rumination (Joormann, 2010). Moreover, this finding suggests that neuropsychological working memory processes (e.g., working memory capacity and manipulation) as well as the content of working memory (e.g., negative automatic thoughts) are associated with depressive symptoms in adolescence, given that the meta-analysis was based on studies using performance-based neuropsychological tasks. Although, it is unclear if this relationship is also present for visuospatial working memory and depressive symptoms as a meta-analysis was not performed regarding this domain of working memory. Again, working memory findings are undermined by the generally poor quality and high heterogeneity of the source studies. Additionally, findings may be of limited clinical significance given the small (but statistically significant) effect size.

Convincing meta-analytic evidence was not found in favour of an association between cognitive flexibility measured by perseverative errors and depressive symptoms. Nor was there systematic review evidence in favour of an association between cognitive flexibility and depressive symptoms when considering other measures of cognitive flexibility. These findings fail to support the notion that cognitive flexibility is an influence on depressive symptoms in adolescence, as predicted by the shifting account of rumination (Koster et al., 2011). Taken together with evidence in favour of small but statistically significant associations between cognitive inhibition of distraction and depressive symptoms and working memory and depressive symptoms, these findings suggest that cognitive inhibition
of distraction and auditory-verbal working memory but not cognitive flexibility are associated with depressive symptoms in adolescence.

There was some evidence, of limited quality, in the systematic review for small associations between aspects of “hot” cognitive control and depressive symptoms in adolescence. Specifically, one study found evidence of a negative association between attentional control in the context of emotional faces and depressive symptoms in adolescents exposed to maltreatment but not those who were not (Gray et al., 2016). This finding suggests an attentional bias away from angry expressions is associated with depressive symptoms in maltreated youths. By contrast, a study using a facial GNG task reported moderately strong associations between facial GNG performance and depressive symptoms, which suggests a bias towards negative emotions (Han et al., 2012). This discrepancy might have arisen because different “hot” cognitive control abilities were considered in each study. The former study probed “hot” attentional control while the latter study measured “hot” behavioural inhibition, although the authors interpreted this finding in the context of “hot” attentional control. Alternatively, the discrepancy could reflect clinical heterogeneity across studies. The first study recruited adolescents in care who were or were not exposed to maltreatment, but did not consider diagnostic thresholds for depression, while the association in the latter study was observed amongst adolescents with diagnosis of depression. Another study reported a negative correlation between low “hot” cognitive flexibility and low depressive symptoms (Stewart et al., 2018). Counterintuitively, this finding suggests that adolescents who were able to switch their focus between angry and neutral faces more quickly displayed more depressive symptoms. However, this association was small and no longer present at a follow up, potentially indicating that the concurrent association was not robust.
Ultimately, findings linking aspects of “hot” cognitive control and depressive symptoms should not be overinterpreted as they arise from single studies. The associations may be specific to the samples, such as adolescents who have experienced maltreatment (i.e., in Gray et al., 2016) or who meet diagnostic criteria for depression (i.e., in Han et al., 2012). Overall, the systematic review of “hot” cognitive control and depressive symptoms indicate that there might not be a universal relationship between “hot” cognitive control and depressive symptoms in adolescence, even though various aspects of “hot” cognitive control share overlapping neural substrates (Poletti, 2009).

It is important to consider that the source studies across meta-analyses and the systematic review were generally of poor overall quality. A major limitation across studies was inadequate sample size justification which resulted in a lack of clarity regarding statistical power to reliably detect small effects. Another common limitation was insufficient consideration of the reliability of measures, which again makes it harder to distinguish genuine effects from measurement error. There was also substantial clinical, methodological, and statistical heterogeneity across studies. Clinically, studies included various samples including those with and without a diagnosis of depression. Methodologically, the studies used various tasks to measure the same cognitive control constructs. Differing measures of depressive symptoms were also used, including adolescent self-reported scales and clinician-rated diagnostic schedules. Statistically, there was evidence of considerable heterogeneity in most of the meta-analyses performed, except for the meta-analysis regarding the inhibition of overlearned responses. Future research should ensure that statistical power and the reliability of measures are properly established to improve the quality of studies in the field. Future studies could also harmonise the measures of cognitive control and depressive symptoms used across studies to reduce methodological heterogeneity.
Relevant covariates, which it was not possible to consider in this systematic review and meta-analysis, include diagnostic status for depression and levels of other related internalising constructs. Regarding diagnostic status for depression, it is possible that adolescents who met clinical criteria may have displayed more pronounced associations (i.e., stronger correlation coefficients) between cognitive control and depressive symptoms than those who did not. It is important to note that adopting a continuum approach to adolescent depression does not necessarily preclude consideration of categories (i.e., “depressed” versus “nondepressed”). Neither does employing a categorical approach automatically prevent consideration of continuous symptoms. Instead, adolescents meeting clinical criteria for a diagnosis of depression can be situated at the far end of the distribution of depressive symptoms in adolescence. Unfortunately, only two of the source studies reported associations between cognitive control and depressive symptoms in adolescents with a depression diagnosis (Han et al., 2012; Peters et al., 2019). Neither of these studies reported separate associations between cognitive control and depressive symptoms in adolescents who did not cross the clinical threshold, which prevented meaningful consideration of this covariate. All other source studies which sampled adolescents who met diagnostic criteria for mood disorders only reported correlations for the entire sample, which included adolescents who did not meet clinical criteria (Dickson et al., 2017; Jandrić et al., 2021; Kavanaugh et al., 2012; Valentino et al., 2012; Wagner et al., 2015b). Regarding relevant internalising constructs (e.g., anxiety), it is possible that these influenced relationships between cognitive control and depression because all aspects of internalising are highly comorbid in adolescence (Essau & de la Torre-Luque, 2021). Only three studies measured anxiety (Gray et al., 2016; Han et al., 2016), one study measured social anxiety (Morea & Calvete, 2021), and one study measured post-traumatic stress disorder (Kim et al., 2021) as well as depressive symptoms. None of these studies statistically controlled for these symptoms when
calculating associations between cognitive control and depressive symptoms. Accordingly, it was not possible to meaningfully consider the impact of other internalising problems. Careful consideration of both diagnostic status and levels of related internalising problems is warranted in future research.

Another unexplored possibility is that there might be an indirect relationship between cognitive control and depressive symptoms which is influenced by another cognitive variable or variables. Two potential variables are over-general memory (OGM) and problem-solving ability. Over-general memory is characterised by difficulty with retrieving specific autobiographical memories (Williams & Broadbent, 1986). It is a feature of depression in adolescence as well as adulthood (Kuyken & Dalgleish, 2011; Stange et al., 2013). The CaR-FA-X model (Williams et al., 2007) theorises that issues with cognitive control, amongst other difficulties, can result in OGM which undermines problem solving ability and leads to depressive symptoms. Cognitive control can also directly influence problem solving ability, without mediation by OGM, in the model. In both routes to depression, the relationship between cognitive control and depressive symptoms depends on other intermediary variables. This may account for the generally small bivariate associations between aspects of cognitive control and depressive symptoms observed in this systematic review and meta-analysis.

This systematic review and meta-analysis features at least three limitations which may undermine the reliability and validity of its findings. First, inclusion criteria for study samples were broad, permitting adolescents both with and without clinical depression. The review and meta-analysis deliberately prioritised individual differences in line with a dimensional approach to depressive symptoms in adolescence. However, it is possible that associations between cognitive control and depressive symptoms are only present in adolescents who meet clinical criteria for a diagnosis of depression. Indeed, systematic reviews and meta-analyses that adopted a categorical approach to depression generally, but not universally,
report evidence of cognitive control impairments in adolescents with clinical depression relative to adolescents without depression (Baune et al., 2014; Goodall et al., 2018; Vilgis et al., 2015; Wagner et al., 2015a). Only two of the reviewed studies reported correlations between cognitive control and depressive symptoms in adolescents with a diagnosis of depression (Han et al., 2012; Peters et al., 2019) and neither of these studies reported equivalent correlations for controls, prohibiting consideration of diagnostic effects in the present meta-analyses.

Second, the review adopted a synthesis strategy based on specific cognitive control constructs (e.g., inhibition of automatic responses, inhibition of distraction, behavioural inhibition, etc.) rather than more general domains (e.g., inhibition). This is defensible given the need to examine partially distinguishable aspects of cognitive control, as implicated in theoretical models relevant to adolescent depression adults (Joormann, 2010; Koster et al., 2011). However, this approach may have neglected potential associations between depressive symptoms and the elements of cognitive control that is shared (Miyake et al., 2000; Miyake & Friedman, 2012). Although, speculatively, this seems unlikely given the preponderance of non-significant correlation coefficients in the reviewed studies.

Finally, while the systematic review and meta-analyses focused its inclusion/exclusion criteria on tightly controlling the nature of cognitive control tasks, considerable heterogeneity was tolerated on measures of depression. In terms of the format of these instruments, they included self-report, parent-reported, and clinician-rated formats. Similarly, regarding the content of these measures, both measures which are symptom counts aligned to diagnostic criteria (e.g., DISC-IV) and measures aligned to theoretical models of depression (e.g., BDI-II) were included. While the reliability and validity of all these measures is established, it is possible that they capture qualitatively different aspects of depressive symptomatology (Fried, 2017). Subsequently, the heterogeneity of depression
measures across the included studies may have obscured associations between cognitive control and depressive symptoms.

The systematic review and meta-analyses has several implications for clinical practice and future research. First and foremost, the findings have implications for clinical models of depression. The findings were broadly consistent with the inhibition-working memory (Joorman, 2010) and working memory-based (Whitmer & Gotlib, 2013) models of depression, which suggests that these adult-based models may also apply to adolescents. Moreover, the findings indicate that the inhibition-working memory model (Joorman, 2010) could be refined to focus on the inhibition of distracting material, rather than inhibition more generally (i.e., including inhibition of distraction, inhibition of overlearned material, behavioural inhibition, etc.), as a maintenance factor for depressive symptoms in adolescence. In contrast, convincing evidence was not found in favour of the cognitive flexibility/switching failure model of depression (Koster et al., 2011), suggesting that this model may not apply in adolescence.

Second, regarding the assessment of adolescents with depression, the review suggests that cognitive control (namely inhibition of distracting information, auditory-verbal working memory, and “hot” cognitive control) should be assessed alongside depressive symptoms when adolescents present with depression. Third, regarding the treatment of adolescents with depression, the review suggests that cognitive remediation targeting the inhibition of distracting information, auditory-verbal working memory, and “hot” cognitive control may be a useful adjunct to established cognitive-behavioural treatments. Indeed, there is some preliminary evidence that cognitive training targeting the executive (i.e., cognitive inhibition) components of performance on n-back working memory tasks is associated with reductions in sub-clinical depressive symptoms in adolescents (Beloe & Derakshan, 2019). The present findings suggest that focusing on the inhibition of distracting information and “hot” cognitive
control might result in incremental therapeutic gains, which could potentially have a large impact on development.

Fourth, the findings may help to elucidate the active mechanisms in mindfulness interventions, which are an effective treatment for depression in adolescence (Reangsing et al., 2020). For example, mindfulness may reduce depressive symptoms by improving adolescents’ ability to resist distraction (i.e., cognitive inhibition of distracting information), maintain a focus on the present moment (i.e., working memory), and being able to focus despite strong emotions (i.e., “hot” attentional control). Indeed, there is some evidence that mindfulness enhances the inhibition of irrelevant information in adolescents (Sanger & Dorjee, 2016).

Finally, by drawing attention to continuously measured depressive symptoms and executive functioning abilities as potentially important areas of variation in adolescence, this systematic review and meta-analysis might have implications for future research practices. Explicitly considering the continuum of depressive symptoms in adolescents does not exclude adolescents meeting, or not meeting, diagnostic criteria for depression. Additionally, highlighting individual differences, rather than average diagnostic group differences, on neuropsychological tasks may provide more granular information regarding executive functioning abilities, without masking potential deficits by excluding adolescents who perform at unusually low levels. The continuum approach employed in this systematic review and meta-analysis might be fruitful for understanding the interplay between cognitive functioning and various psychological issues in adolescence and beyond.
Conclusion

In summary, the systematic review and meta-analyses suggest that individual differences in resisting the effects of distracting information (cognitive inhibition) and difficulty maintaining and manipulating information held in the mind’s eye (working memory) are associated with depressive symptoms in adolescence. These meta-analytic findings provide some support for the applicability of the inhibition-working memory (Joorman, 2010) and working memory-based (Whitmer & Gotlib, 2013) accounts of depressive symptoms in adolescence, respectively. However, it should be noted that the associations between aspects of executive functioning and depressive symptoms were small and may not be of marked clinical significance. There was also mixed systematic review evidence in favour of associations between aspects of “hot” cognitive control and depressive symptoms in adolescence, although the exact nature of these relationships is unclear. Unfortunately, the literature reviewed was generally of poor quality. Future studies should ensure adequate statistical power and consider the reliability of measures so that greater confidence can be placed in typically small effects. Additionally, researchers could consider using the same measures of cognitive control and depressive symptoms to reduce methodological heterogeneity.
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Disentangling the Relationships Between Motor Control and Cognitive Control in Young Children with Symptoms of ADHD

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Abstract

Objective: Children with ADHD experience difficulties with motor and cognitive control. However, the relationship is poorly understood. As a step towards improving treatment, this study investigated associations between specific aspects of motor control and cognitive control in children with varying levels of hyperactive-impulsive symptoms. Method: A heterogeneous sample of 255 children of 4 to 10 years of age (median = 6.50, MAD = 1.36) completed a battery of tests probing motor generation, visuomotor fluency, visuomotor flexibility, cognitive inhibition, verbal and visuospatial working memory, and cognitive flexibility. Their carers were interviewed regarding their hyperactive-impulsive symptoms. Approximately 26% of the analysed sample met diagnostic criteria for ADHD. Multiple linear regression analysis was used to determine whether specific aspects of motor control were associated with specific aspects of cognitive control, and whether any associations were moderated by hyperactive-impulsive symptoms. Additionally, cognitive modelling (the drift diffusion model approximated with EZ-DM) was used to understand performance on a cognitive inhibition task. Results: Visuomotor fluency was significantly associated with cognitive inhibition. Visuomotor flexibility was significantly associated with cognitive flexibility. There were no significant moderation effects. Cognitive modelling was inconclusive. Conclusions: The ability to fluently perform visually guided continuous movement is linked with the ability to inhibit the effects of distracting information. The ability to spontaneously use visual information to flexibly alter motor responses is related to the ability to cognitively shift from one frame of mind to another. These relationships appear to be quantitatively and qualitatively similar across the childhood hyperactive-impulsive continuum.
**Introduction**

Children with attention deficit hyperactivity disorder (ADHD) experience difficulties with controlling movement and controlling thought. In addition to hyperactivity, which is a core feature of hyperactive-impulsive and combined ADHD (American Psychiatric Association, 2013; World Health Organization, 2019), children with ADHD can experience several other motor difficulties (see Kaiser et al., 2015, for a review). These include challenges with fine motor skills (e.g., Mokobane et al., 2019; Polderman et al., 2011), motor timing (e.g., van der Meer et al., 2016; Rosch et al., 2013; Rubia et al., 2003; Zelaznik et al., 2012), motor overflow (e.g., Denckla & Rudel, 1978; Mostofsky et al., 2003), motor generation (e.g., Rommelse et al., 2008), and visuomotor control (e.g., Fabio et al., 2022; Tirosh et al., 2006). Typically, children with ADHD display increased response variability and reaction time variability as well as decreased overall accuracy of motor functioning (e.g., Demers et al., 2013; Kalff et al., 2005; Rommelse et al., 2008). Cognitively, children with ADHD often have difficulties with inhibition, working memory, cognitive flexibility, planning and organisation as well as in overall ability (Pievsky & McGrath, 2017). Cognitive control in childhood is predictive of educational, occupational, and health outcomes in adulthood (Moffit et al., 2011). Subsequently, it is important to identify and capitalise on opportunities for early intervention. Improving our understanding of the relationship between motor control and cognitive control has the potential to inform early interventions.

**The Relationship between Motor Control and Cognitive Control**

The growth of cognitive control system is entwined with the refinement of the motor system in typical development (Diamond, 2000; van der Fels et al., 2014). Faster (slower) acquisition of motor milestones is strongly predictive of greater (poorer) cognitive control
abilities in adulthood (Murray et al., 2006; 2007; Ridler et al., 2006). While motor and cognitive issues co-occur in childhood ADHD, the potential relationships between them are poorly understood. Koziol et al. (2013) argue that motor and cognitive difficulties co-occur in childhood ADHD because of abnormal functioning in overlapping neural substrates. It is known that frontostriatal (Diamond, 2013; Frank & Badre, 2015) and corticocerebellar (Bellebaum & Daum, 2007; Blaedel & Bracha, 1997; Diamond, 2000; Ramnani, 2006) circuits are important for the control of movement as well as the control of thought (Koziol et al., 2012, 2014; Middleton & Strick, 2000). Indeed, childhood ADHD is associated with delayed maturation in these structures as well as in the prefrontal cortex (Sharma & Couture, 2014). As the development of motor control begins before the development of cognitive control (Njiokiktjien, 2007; Piek et al., 2008), difficulties in motor control may underly difficulties in cognitive control (Koziol et al., 2013). Treating motor difficulties might therefore benefit cognitive control and life outcomes for children with symptoms of ADHD. Indeed, research that clarifies the relationship between cognitive and motor control may be helpful for developing more effective and informed targets for cognitive remediation interventions (e.g., Meyer et al. 2020; Pauli-Pott et al., 2021).

For children without ADHD, performance on various tasks involving motor control is associated with cognitive inhibition (Livesey et al., 2006; Rigoli et al., 2012; Stöckel & Hughes, 2016), working memory (Stöckel & Hughes, 2016; Rigoli et al., 2012; Wassenberg et al., 2005), and cognitive flexibility (Fang et al., 2017) in some but not all studies. For children with ADHD, one study suggests that motor control is most consistently associated with cognitive inhibition (Tseng et al., 2004). Together, these studies suggest that there is not a general relationship between motor control and cognitive control, which implies that efforts should be focused on understanding associations between specific abilities. Unfortunately, associations between motor control and specific aspects of cognitive control
are not well replicated across existing studies. The most consistent finding is that motor control and cognitive inhibition are related (e.g., Livesey et al., 2006; Stöckel & Hughes, 2016; Rigoli et al., 2012), but evidence for associations with other aspects of cognitive control should not be dismissed.

**Motor Control Skills**

One factor that makes it difficult to understand the relationship between motor and cognitive control is that they are complex, multifaceted constructs. Many existing studies assessed general motor competence and motor-related activities (e.g., running, throwing, and catching). Motor control can refer to a wide array of underlying abilities, including motor generation, visuomotor fluency, and visuomotor flexibility (de Sonneville, 2011; Njiokiktjien, 2007). *Motor generation* refers to the ability to voluntarily generate consistent motor output over time, such as tapping with one’s finger for a prolonged period. Motor generation is important for initiating and continuing to perform practically all tasks with a physical element. It can therefore be considered a foundational motor ability. *Visuomotor fluency* involves controlling movement in relation to unchanging visual stimuli, such as tracing a circle. Visuomotor fluency is important for writing between or colouring within the lines, for example. It is a less demanding skill than *visuomotor flexibility*, which involves controlling movement in unpredictable visual situations, such as when a target to be followed moves in an unexpected way. Visuomotor flexibility is likely important for playing computer games and taking part in sport where children must visually track moving stimuli (e.g., a football) and alter their movement (e.g., the motion of their feet) accordingly.
Cognitive Control Skills

Similarly, cognitive control is an umbrella term which can be separated into three core components in young children. These are cognitive inhibition, which involves withholding automatic responses and/or resisting the effect of distracting information; working memory, which involves holding and manipulating information temporarily held in the mind; and cognitive flexibility, which involves switching between different frames of mind (Henry & Bettenay, 2010; Miyake et al., 2000; Miyake & Friedman, 2012). These variables have been identified as three separable constructs in factor analytic studies in children (Anderson, 2010; Fisk & Sharp, 2004; Garon et al., 2008; Henry & Bettenay, 2010; Lehto et al., 2003). Previous relevant studies have fractionated cognitive control along these lines (e.g., Rigoli et al., 2012), but they have generally not also considered specific aspects of motor control.

Associations between Specific Aspects of Motor Control and Cognitive Control

Understanding of links between specific motor control and cognitive control skills can inform the development of motor and cognitive remediation programmes by identifying which specific functions should be targeted in early childhood. As an initial effort towards this goal, we tested plausible hypotheses about associations between specific aspects of motor control (motor generation, visuomotor fluency, and visuomotor flexibility) and cognitive control (cognitive inhibition, working memory, and cognitive flexibility). Due to the lack of previous research, these hypotheses were developed through considering limited prior work in this area, logical reasoning, and necessary speculation (Swedberg, 2021). Our hypotheses were pre-registered (https://osf.io/nphb9) prior to analysis. We summarise our rationale for our hypotheses below.
**Motor Control and Cognitive Inhibition**

Being able to produce sufficient motor output (motor generation) and fluidly perform visually guided movement in predictable situations (visuomotor fluency) may be necessary to respond quickly and accurately in visual situations involving cognitive inhibition (Rigoli et al., 2012). Accordingly, we hypothesised that motor generation and visuomotor fluency would be positively associated with cognitive inhibition. (We did not hypothesise that visuomotor flexibility would be associated with cognitive inhibition because cognitive inhibition is often invoked in fast-paced tasks, which do not provide sufficient time for flexible cognition.)

**Motor Control and Working Memory**

Because motor generation involves maintaining persistent motor output over time (de Sonneville, 2011; Njioiktjien, 2007) and working memory involves the maintenance of information over time (Baddeley, 2012; Barrouillet & Camos, 2007), we hypothesised that motor generation would be positively associated with working memory. Additionally, because being able to continually adjust visually guided movements in predictable (visuomotor fluency) and unpredictable (visuomotor flexibility) settings is akin to manipulating information held in the mind’s eye in response to persistent and changeable environmental demands (working memory manipulation), we hypothesised that visuomotor fluency and visuomotor flexibility would be positively associated with working memory manipulation.

**Motor Control and Cognitive Flexibility**

To switch between different frames of mind, cognitive flexibility depends on the cognitive inhibition of distracting information and the maintenance and manipulation of
information in working memory (Cragg & Chevalier, 2012). We already hypothesised that motor generation would be positively associated with cognitive inhibition and working memory, and that visuomotor fluency and visuomotor flexibility would be positively associated with working memory manipulation. Additionally, both visuomotor flexibility and cognitive flexibility involve adaptation to unpredictable changes in the environment, albeit in different domains. Subsequently, we hypothesised that motor generation, visuomotor fluency, and visuomotor flexibility would be positively associated with cognitive flexibility.

**The Moderating Effect of Hyperactive-Impulsive Symptoms**

Sergeant (2000, 2005) theorised that activation (i.e., physiological readiness to respond) can influence cognitive processing. Specifically, both too much and too little physiological activation can undermine cognition, and hence there is an optimal window to support cognitive task performance for children with ADHD. Therefore, because children with high levels of hyperactive-impulsive symptoms can exhibit too much activation (e.g., Burley et al., 2021; Murillo et al., 2015), we hypothesised that greater motor generation in children with higher levels of hyperactive-impulsive symptoms would be associated with poorer cognitive control skills. Specifically, any associations between motor generation and cognitive inhibition, working memory, and cognitive flexibility would be moderated by hyperactive-impulsive symptoms by changing the positive sign of the associations to negative for children at the high end of the hyperactive-impulsive continuum (Hayes, 2017).

Additionally, we speculatively hypothesised that children with higher hyperactive-impulsive symptoms and poorer visuomotor fluency and visuomotor flexibility would display even poorer working memory manipulation. In other words, positive associations between visuomotor fluency and visuomotor flexibility and working memory manipulation
would be moderated (Hayes, 2017) by hyperactive-impulsive symptoms through increasing the strength of the aforementioned association.

**A Process Approach to Understanding Cognitive Control**

Another barrier to understanding links between specific aspects of motor and cognitive control is that tests of cognitive inhibition, working memory, and cognitive flexibility are not-process specific, despite purporting to measure a particular element of cognitive control. For example, regarding cognitive inhibition, a child’s performance on a flanker task (in which they must quickly select a target stimulus that is flanked by either congruent or incongruent stimuli on either side) can depend on how efficiently they process information, whether they prioritise speed or accuracy, and how long it takes them to encode stimuli and prepare for motor actions (Ratcliff & McKoon, 2008).

By separating overall performance into subcomponents, cognitive modelling can highlight specific difficulties and qualitatively different cognitive approaches (e.g., a speed-accuracy trade-off). Cognitive modelling can help us move beyond a deficit approach, which focuses solely on what is wrong, to a process approach (e.g., Bernstein, 2013), which clarifies why children are struggling. Knowing how children achieve a score (e.g., by prioritising accuracy over speed) as well as what they score compared to normative data, could improve understanding of their strengths and difficulties and inspire personalised treatment plans. Also, as cognitive modelling facilitates an appreciation of individual differences, its use is consistent with contemporary dimensional approaches to understanding ADHD symptoms in childhood (e.g., Musser & Raiker, 2019).

Several studies have used the Drift Diffusion Model (DDM; Ratcliff & McKoon, 2008), which models processing efficiency, the speed-accuracy trade-off, and stimuli encoding and motor response execution time, to understand cognitive differences in children.
with ADHD. Generally, studies suggest that children with ADHD process information less efficiently than their typically developing peers (Haller et al., 2021; Huang-Pollock et al., 2017, 2020; Karalunas et al., 2012). Studies have generally not found evidence for group differences in the speed-accuracy trade-off (Feldman & Huang-Pollock, 2021; Haller et al., 2021; Karalunas et al., 2012) or for associations between continuously measured ADHD symptoms and the speed-accuracy trade-off (Feldman & Huang-Pollock, 2021); although, one study found evidence of increased caution in a group of children with ADHD (Fosco et al., 2019). Evidence for differences in stimuli encoding and motor response execution time is mixed. One study reported that children with ADHD take less time to encode stimuli and prepare and execute motor responses (Metin et al., 2013) while other studies did not report any differences (Fosco et al., 2019; Karalunas et al., 2012). Overall, these findings are equivocal. This may be because the studies used a variety of tasks probing various cognitive and perceptual abilities. It is unclear whether DDM parameters are best considered task-invariant latent constructs (Schmiedek et al., 2007) or whether differences in them arise from differing task demands (Koziol, 2014). In the current study, we focused our cognitive modelling on flanker task performance as a prototypical measure of cognitive inhibition (Zelazo et al., 2013), which, in comparison with other aspects of cognitive control, has been more frequently linked with motor control in children with and without ADHD (Livesey et al., 2006; Stöckel & Hughes, 2016; Rigoli et al., 2012).

Previous studies have not considered whether the processing efficiency, speed-accuracy trade-off, and time for encoding stimuli and motor response execution underlying cognitive inhibition are influenced by motor control abilities and moderated by hyperactive-impulsive symptoms. We tentatively hypothesised that motor generation would be positively associated with stimuli encoding and motor response time underlying cognitive inhibition, as at face value both involve the execution of motor actions. We also hypothesised that
visuomotor fluency would be associated with the speed-accuracy trade-off, but we did not make a directional hypothesis because children who are better able to control their movement in response to visual stimuli could feasibly show more liberal (i.e., prioritising speed) or more conservative (i.e., prioritising accuracy) approaches in the speed-accuracy trade-off underlying cognitive inhibition. Additionally, we hypothesised that motor generation and visuomotor fluency would be positively associated with processing efficiency, because the ability to generate consistent, prolonged motor output and to adjust movement in response to consistent visual information might lead to increased processing efficiency underlying cognitive control. Finally, we explored whether these associations would be moderated by hyperactive-impulsive symptoms, but we did not make any specific hypotheses in this domain.

The Current Study

In summary, childhood ADHD involves difficulties with both motor and cognitive control. Indeed, motor control difficulties may contribute to cognitive control difficulties. However, current findings are equivocal. A key challenge is that motor control and cognitive control encompass several skills. To better understand and treat children’s difficulties, it is important to clarify the relationship between motor control and cognitive control. The primary aims of our study were to investigate which aspects of motor control (motor generation, visuomotor fluency, and visuomotor flexibility) are associated with which aspects of cognitive control (cognitive inhibition, working memory, and cognitive flexibility) and whether these relationships are moderated by hyperactive-impulsive symptoms. We also used cognitive modelling to indicate how motor control might influence cognitive inhibition in terms of processing efficiency, the speed-accuracy trade-off, and encoding of stimuli and motor execution time underlying cognitive inhibition. We
anticipated that our study would provide foundational knowledge, which may highlight potential avenues for early intervention for children with motor and cognitive issues, such as cognitive remediation programmes. To summarise, our hypotheses were as follows:

**Part A: Hypotheses Regarding Associations Between Specific Aspects of Motor Control and Cognitive Control and their Moderation by Hyperactive-Impulsive Symptoms:**

1. Motor generation and visuomotor fluency would be positively associated with cognitive inhibition.

2. Motor generation would be positively associated with working memory.

3. Visuomotor fluency and visuomotor flexibility would be positively associated with working memory manipulation.

4. Motor generation, visuomotor fluency, and visuomotor flexibility would be associated with cognitive flexibility.

5. Greater motor generation in children with higher levels of hyperactive-impulsive symptoms would be associated with poorer cognitive inhibition, working memory, and cognitive flexibility.

6. Children with higher hyperactive-impulsive symptoms and poorer visuomotor fluency and visuomotor flexibility would display even poorer working memory manipulation.

**Part B: Hypotheses Regarding Specific Processes Underlying Cognitive Inhibition:**

1. Motor generation would be positively associated with stimuli encoding and motor response execution time underlying cognitive inhibition.

2. Visuomotor fluency would be associated with the speed-accuracy trade-off underling cognitive inhibition.
3. Motor generation and visuomotor fluency would be positively associated with processing efficiency underlying cognitive inhibition.

We were also interested in whether/how these potential associations were moderated by hyperactive-impulsive symptoms but did not make any directional hypotheses.
Methods

Our hypotheses, methods, and analyses were pre-registered after data collection had begun: https://osf.io/nphb9. Our methods section is a near reproduction of our preregistration document (see Appendix F). Deviations from the pre-registered analysis plan are stated below.

Participants

Recruitment and Sample

Data were collected from 399 children between 4 to 10 years of age who were referred to the Neurodevelopmental Assessment Unit at Cardiff University. Ethical approval was gained from the University (EC.16.10.11.4592GRA5; see Appendix G). Children were referred for various internalising and externalising problems. Recruitment was from schools across South Wales who referred children for assessment with parental consent (Appendix H). The referrer received a report describing the child’s strengths/difficulties on a selection of the normative tasks used alongside recommended compensatory strategies (the reports were overseen by an Educational Psychologist). Data for 255 children were available following the exclusion of missing data. Children in this sample were 6.5 years old on average (SD = 1.05). Approximately 31% of this sample were female and 64% male; sex data were unavailable for 5% participants. 16 children (6.27%) met conservative diagnostic criteria for hyperactive-impulsive ADHD as assessed by with Development and Wellbeing Assessment. 4.31% met criteria for inattentive ADHD and 25.88% met criteria for combined ADHD. Data for a subset of 150 children were used for cognitive modelling. Approximately, 31% this subsample were female and 68% were male; sex data were unavailable for 0.7% participants. In the subsample, 2.7% children met criteria for hyperactive-impulsive ADHD, 4.0% met criteria for inattentive ADHD, and 12% met
criteria for combined ADHD. Further characteristics of the sample(s) are summarised in Table 2.

**Inclusion/Exclusion Criteria**

It was intended that children would be excluded if they had estimated general cognitive functioning below a scaled score of 70 (where M = 100, SD = 15) on the Lucid Ability Test (Singelton, 2001), to ensure that individual differences in motor and cognitive control ability were investigated, rather than the effects of very low general cognitive ability and possible intellectual disability. However, no children scored below this criterion.

**Power Analysis**

An a priori power calculation using G*Power (Faul et al., 2009) indicated that a sample of at least 153 children was needed to confer at least 80% power to detect a relatively small relationship of $f^2 = 0.065$ between motor control and cognitive control, given the inclusion of seven predictors in a linear multiple regression model with $R^2$ increase. The anticipated effect size was selected from Rigoli et al. (2012) who observed that motor control significantly predicted a small portion of the variance (equivalent to $f^2 = 0.065$) on a test of inhibition in a sample of adolescents.

**Measures**

**Motor Control**

**Motor Generation.** The Amsterdam Neuropsychological Tasks (ANT; de Sonneville, 1999) Tapping task is a measure of self-generated motor output without internal or external cues (Rommelse et al., 2008). Children must click a computer mouse button with their dominant hand as many times as possible within a 60-second time limit. The task was
validated in a convenience sample of 913 children (de Sonneville, 2011). The task generates a z-score for the number of taps generated, which is referenced to an age-stratified normative sample. Tapping shows high test-retest reliability in children (Njiokiktjien, 2007, p. 195).

**Visuomotor Fluency.** ANT Tracking is a test of visuomotor fluency (Slaats-Willemse et al., 2005). Children must trace a circle with a computer mouse and cursor with their dominant hand. Thus, movement follows a predefined trajectory during the task. Validity was established in a convenience sample of 1789 children (de Sonneville, 2011). Tracking provides norm-referenced z-scores for accuracy (i.e., the mean distance from the midline averaged across equal-sized segments of the circle) and variability (i.e., the standard deviation of the mean distance from the midline averaged across equal-sized segments of the circle) of movement. As accuracy and variability were very strongly related (r = 0.91), only the z-scores for accuracy were included in statistical models to guard against multicollinearity. Scores were reversed so that higher values represented better performance.

**Visuomotor Flexibility.** ANT Pursuit is a test of visuomotor flexibility (Slaats-Willemse et al., 2005). Children must follow an on-screen target, which moves in an unpredictable manner, with a computer mouse and a cursor. Thus, movement during the task is spontaneous. Validity was established in a convenience sample of 1789 children (de Sonneville, 2011). Pursuit also provides norm-referenced z-scores for accuracy (i.e., the mean distance from the trajectory of a target moving in an unpredictable manner) and variability (i.e., the standard deviation of the mean distance from the trajectory of a target moving in an unpredictable manner) of movement. However, due to a very strong correlation between accuracy and variability (r = 0.90), only the accuracy z-scores were included in models. Scores were reversed so that higher values signified better performance.
**Hyperactive-Impulsive Symptoms**

**Development and Wellbeing Assessment ADHD Hyperactivity Symptom Score.**

The Hyperactivity score from the Development and Well-Being Assessment (DAWBA) Attention and Activity scale was used as a measure of hyperactive-impulsive ADHD symptoms (Goodman et al., 2000). The DAWBA is a structured interview with a parent as the informant. The score was entered as covariate in the regression analyses described below.

**Cognitive Control**

**Cognitive Inhibition.** The National Institute of Health (NIH) Toolbox Flanker is a test of cognitive inhibition (Zelazo et al., 2013). Children must selectively attend to a central target stimulus while inhibiting attention to laterally placed stimuli. Children aged 3-7 years old are initially presented with 20 trials of fish stimuli (12 congruent, 8 incongruent). If a child aged 3-7 scores ≥ 90% on the fish stimuli, 20 additional trials with arrows are presented (12 congruent, 8 incongruent). Children aged 8+ are presented with 20 trials of arrow stimuli (12 congruent, 8 incongruent). The task provides a single combined score for accuracy and, for participants who achieve more than 80% accuracy, reaction times. This score is age-corrected by reference to normative data. Test-retest reliability is .92 (Zelazo et al., 2013). Individual trial data for accuracy and reaction time were used for cognitive modelling (see below).

**Cognitive Flexibility.** The NIH Toolbox Dimensional Change Card Sort (DCCS) task is a test of cognitive flexibility (Zelazo, 2006). Children must sort a series of cards according to one rule (by colour or shape) before this rule changes and they must sort the series of cards according to a new rule. The DCCS task provides a single combined score for accuracy and, for participants who achieve more than 80% accuracy, reaction times. This
score is age-corrected by reference to normative data stratified by year of age. Test-retest reliability is .92 (Zelazo et al., 2013).

**Verbal Working Memory.** The Automated Working Memory Assessment (AWMA) Backwards Digit Recall is a test of verbal working memory (Alloway et al., 2006). Children hear a sequence of digits, which increases in length on subsequent trials, and must recall the numbers in backwards order. The score is age-corrected by reference to normative data stratified by year of age. Test-retest reliability is .64 (Alloway et al., 2008).

**Visuospatial Working Memory.** AWMA Mister X is a test of visuospatial working memory (Alloway et al., 2006). Children are presented with two figures with different coloured hats who are holding a ball in one of two hands. One of these figures is rotated. Children are asked whether the two figures are holding the ball in the same or different hands and then to recall where the figure with the blue hat was holding the ball. Two metrics are generated, an accuracy score (which reflects foundational visuospatial working memory abilities such as capacity and maintenance) and a processing score (which reflects the manipulation aspect of visuospatial working memory). Each measure is expressed as a standard score (M = 100, SD = 15) which is age-corrected by reference to normative data. Test-retest reliability is .77 (Alloway et al., 2006).

**Statistical Analyses**

**Multiple Linear Regression Analyses**

Several multiple linear regression analyses were used to examine individual differences in motor control, cognitive control, and hyperactive-impulsive ADHD symptoms. The model terms were pre-registered in accordance with the study hypotheses. Post-hoc simple slope analyses were also planned to understand any moderation effects, but these were not necessary (as all moderation terms were non-significant). Children were excluded from
an analysis if they were missing data for the variables included in that analysis. A data
imputation strategy was not used because due to the presence of developmental difficulties in
the sample data were unlikely to be missing completely at random. Two outliers were
removed from Tracking task data (z scores of -22.4 and -22.5) and a single outlier was
removed from data for the Pursuit (a z score of – 63.0) task after inspecting pre-
transformation histograms (see Appendix I). Deviating from the preregistration, data were
transformed using non-paranormal transformation (Liu et al., 2009) prior to analysis to better
meet the assumptions of multiple linear regression. This transformation method maintains
ordinality and therefore preserves the interpretability of variables while meeting model
assumptions (Epskamp & Fried, 2018). Following transformation, data approximated all
assumptions for multiple linear regression analysis (see Appendix J). An overall alpha level
of 0.05 was set as the threshold for statistical significance. Holm-Bonferroni correction was
used when there were multiple models that could each support the same hypothesis (i.e.,
models for Backward Digit Recall and Mister X could both support the hypothesis that motor
generation was associated with working memory). Correction for multiple comparisons was
not performed across all regression models as our inferential models were theoretically
motivated and pre-registered.

Cognitive Modelling

The EZ-DM method (Wagenmakers et al., 2007, 2008) was used to estimate a basic
Drift-Diffusion Model (DDM; Ratcliff & McKoon, 2008) of performance on the NIH
Flanker task for a pragmatically selected subset of 150 children with available trial by trial
data. The DDM assumes that while making a binary decision (e.g., whether to click left or
right on a flanker task), information is continuously sampled, in a noisy diffusion-like
process, from the displayed stimuli array until enough evidence has accumulated to make a
response (Ratcliff & McKoon, 2008). A response occurs once one of two thresholds has been crossed. The accuracy of the response depends on which threshold was hit during the decision process.

The EZ-DM method provides parameter estimates for drift rate, boundary separation, and non-decision time parameters based on mean reaction time, the variance of reaction time, and the percentage of correct responses on the Flanker task. Drift rate is the average slope of the diffusion process and reflects the efficiency with which information is sampled. Boundary separation refers to the distance between the two decision thresholds. Larger values lead to longer decision processes on average, whereas smaller values lead to shorter decision processes on average. A larger (smaller) boundary separation value implies a more conservative (liberal) decision-making style as more (less) evidence is needed for a decision to be made. Non-decision time refers to the time before and after the decision process (which is characterised by drift rate and boundary separation) and reflects the time needed for stimuli encoding and motor response execution (Ratcliff & McKoon, 2008). Congruent and incongruent Flanker trials were modelled in parallel and then the EZ-DM parameter estimates were averaged, giving rise to combined estimates which were statistically analysed.

**Inclusion/Exclusion Criteria for Cognitive Modelling.** Trials featuring non-physiologic anticipation responses (RT ≤ 150ms) were excluded from cognitive modelling (as in Haller et al., 2021). Slow responses of ≥ 3 seconds were also excluded (Ratcliff, 2008).

**Robustness Checks.** Prior to inferential analysis, two checks were performed to ensure that the EZ-DM parameter estimates were robust. First, a parameter recovery routine was used to assess the relative fit of EZ-DM estimates to the empirical data. Second, a comparison of empirical and simulated summary statistics was used to assess the absolute fit
of EZ-DM modelling. Both checks suggested acceptable robustness. Additionally, correlational analysis was used to check whether all EZ-DM parameters were associated with the NIH Flanker score. Full details of these checks are presented in Appendix K. R Markdown code for all analyses is presented in Appendix L.
Results

Descriptive Statistics

Descriptive statistics are presented in Table 2. The descriptive statistics reveal that, on average, children performed within normal limits (+/- 1 SD) on tests of cognitive control and general cognitive ability. This suggests that as, a sample, the children did not display marked cognitive control difficulties. However, children performed considerably poorer on standardised tests of motor functioning, indicating that they experienced difficulties with motor control. As a sample, the children scored particularly low on visuomotor fluency.

Table 2. Descriptive Statistics for the Main Sample and the Cognitive Modeling Subsample.

<table>
<thead>
<tr>
<th></th>
<th>Main sample (N = 255)</th>
<th>Cognitive modeling subsample (n = 150)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (MAD)</td>
</tr>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>6.5 (1.1)</td>
<td>6.50 (1.4)</td>
</tr>
<tr>
<td>IQ (estimated)</td>
<td>98.3 (12.0)</td>
<td>98.0 (11.9)</td>
</tr>
<tr>
<td>ADHD Symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperactive-Impulsive</td>
<td>11.0 (6.7)</td>
<td>13.0 (6.2)</td>
</tr>
<tr>
<td>Inattentive</td>
<td>10.8 (6.0)</td>
<td>12.0 (5.9)</td>
</tr>
<tr>
<td>Motor control tasks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tapping (motor generation)</td>
<td>-1.0 (1.7)</td>
<td>-0.39 (1.1)</td>
</tr>
<tr>
<td>Tracking (visuomotor fluency)</td>
<td>-2.4 (3.3)</td>
<td>-1.40 (2.1)</td>
</tr>
<tr>
<td>Pursuit (visuomotor flexibility)</td>
<td>-2.1 (5.4)</td>
<td>-0.30 (1.7)</td>
</tr>
<tr>
<td>Cognitive control tasks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flanker (cognitive inhibition)</td>
<td>93.0 (14.9)</td>
<td>94.0 (11.9)</td>
</tr>
<tr>
<td>DCCS (cognitive flexibility)</td>
<td>95.5 (14.1)</td>
<td>96.0 (10.4)</td>
</tr>
<tr>
<td>BDR (verbal working memory)</td>
<td>98.7 (16.5)</td>
<td>98.0 (16.3)</td>
</tr>
<tr>
<td>Mr X (visuospatial working memory)</td>
<td>106.1 (17.2)</td>
<td>104.0 (17.8)</td>
</tr>
<tr>
<td>Mr X Processing (working memory manipulation)</td>
<td>103.8 (16.6)</td>
<td>99.0 (13.3)</td>
</tr>
<tr>
<td>Cognitive modeling parameter estimates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drift rate (processing efficiency)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Boundary separation (speed-accuracy trade-off)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nondecision time (stimuli encoding and motor execution)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Note: Statistics are based on untransformed data. ADHD: Attention Deficit Hyperactivity Disorder. BDR: Backwards Digit Recall. DCCS: Dimensional Change Card Sort Task. IQ: Overall cognitive ability estimated with the Lucid Ability Test. MAD: Median absolute deviation. Mr X: Mister X. SD: Standard deviation.
Bivariate Pearson’s correlations amongst variables are displayed in Table 3. In these preliminary analyses, Tracking (visuomotor fluency) and Flanker (cognitive inhibition) performance \( (r = 0.23, 95\% \text{ CI} = 0.11, 0.34, p < .001) \) and Tracking and Dimensional Change Card Sort (cognitive flexibility) performance \( (r = 0.14, 95\% \text{ CI} = 0.02, 0.26, p = .02) \) were significantly correlated. Tracking was also significantly related to Hyperactive-Impulsive Symptoms \( (r = 0.13, 95\% \text{ CI} = 0.00, 0.25, p = .04) \). Additionally, Pursuit (visuomotor flexibility) and Flanker \( (r = 0.27, 95\% \text{ CI} = 0.15, 0.38, p < .001) \), Pursuit and Dimensional Change Card Sort \( (r = 0.14, 95\% \text{ CI} = 0.07, 0.30, p < .001) \), and Pursuit and Backwards Digit Recall (verbal working memory) performance \( (r = 0.21, 95\% \text{ CI} = 0.09, 0.33, p < .001) \) were significantly associated. Hyperactive-Impulsive Symptoms and Mister X (working memory) performance \( (r = 0.37, 95\% \text{ CI} = 0.26, 0.47, p < .001) \) and Hyperactive-Impulsive Symptoms and Mister X Processing (working memory manipulation) performance \( (r = 0.36, 95\% \text{ CI} = 0.25, 0.46, p < .001) \) were significantly correlated. Finally, Tapping (motor generation) was not significantly associated with any cognitive control variable.

**Table 3. Bivariate Correlations amongst Variables.**

<table>
<thead>
<tr>
<th>Contextual variables</th>
<th>Motor control tasks</th>
<th>ADHD symptoms</th>
<th>Cognitive control tasks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>IQ</td>
<td>TP</td>
<td>TR</td>
</tr>
<tr>
<td>IQ</td>
<td>-.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TP</td>
<td>-.11</td>
<td>.14</td>
<td></td>
</tr>
<tr>
<td>TR</td>
<td>.27</td>
<td>.23</td>
<td>.24</td>
</tr>
<tr>
<td>PU</td>
<td>.33</td>
<td>.27</td>
<td>.13</td>
</tr>
<tr>
<td>HI</td>
<td>.26</td>
<td>.07</td>
<td>-.01</td>
</tr>
<tr>
<td>FL</td>
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<td>.26</td>
<td>.00</td>
</tr>
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<td>.12</td>
</tr>
<tr>
<td>BD</td>
<td>-.10</td>
<td>.37</td>
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</tr>
<tr>
<td>MX</td>
<td>-.08</td>
<td>.24</td>
<td>.05</td>
</tr>
<tr>
<td>MX P</td>
<td>-.08</td>
<td>.24</td>
<td>.03</td>
</tr>
</tbody>
</table>

Inferential analyses

All inferential analyses were conducted in accordance with the pre-registered analysis plan. The results for all multiple linear regression analyses are shown in Table 4.

Table 4: Multiple Linear Regression Analyses of Motor Control and Cognitive Control Tasks.

<table>
<thead>
<tr>
<th>Hyp.</th>
<th>Dependent and predictor variables</th>
<th>Adj. $R^2$</th>
<th>$\beta$</th>
<th>SE</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>Flanker (cognitive inhibition)</td>
<td>0.05</td>
<td>-0.00</td>
<td>0.06</td>
<td>-0.01</td>
<td>.99</td>
</tr>
<tr>
<td></td>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping (motor generation)</td>
<td></td>
<td>-0.07</td>
<td>0.07</td>
<td>-1.07</td>
<td>.29</td>
</tr>
<tr>
<td></td>
<td>~ Tracking (visuomotor fluency)</td>
<td>0.21</td>
<td>0.06</td>
<td>3.32</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
<td>0.10</td>
<td>0.06</td>
<td>1.52</td>
<td>.13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping * Hyperactive-Impulsive Sx</td>
<td>0.08</td>
<td>0.07</td>
<td>1.05</td>
<td>.29</td>
<td></td>
</tr>
<tr>
<td>A2</td>
<td>Backwards Digit Recall (verbal working memory)</td>
<td>0.01</td>
<td>0.00</td>
<td>0.04</td>
<td>.96</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>~ Tapping (motor generation)</td>
<td>0.08</td>
<td>0.07</td>
<td>1.17</td>
<td>.24</td>
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<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
<td>0.01</td>
<td>0.06</td>
<td>0.13</td>
<td>.90</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping * Hyperactive-Impulsive Sx</td>
<td>0.12</td>
<td>0.07</td>
<td>1.61</td>
<td>.11</td>
<td></td>
</tr>
<tr>
<td>A2</td>
<td>Mister X (visuospatial working memory)</td>
<td>0.13</td>
<td>-0.00</td>
<td>0.06</td>
<td>-0.03</td>
<td>.98</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping (motor generation)</td>
<td>0.07</td>
<td>0.06</td>
<td>1.06</td>
<td>.29</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
<td>0.38</td>
<td>0.06</td>
<td>6.34</td>
<td>&lt; .001</td>
<td></td>
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<tr>
<td></td>
<td>~ Tapping * Hyperactive-Impulsive Sx</td>
<td>-0.05</td>
<td>0.07</td>
<td>-0.74</td>
<td>.46</td>
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<td>A3</td>
<td>Mister X Processing (working memory manipulation)</td>
<td>0.12</td>
<td>-0.02</td>
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<td>.78</td>
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<td>Intercept</td>
<td></td>
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<tr>
<td></td>
<td>~ Tracking (visuomotor fluency)</td>
<td>-0.04</td>
<td>0.07</td>
<td>-0.57</td>
<td>.57</td>
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<td>~ Pursuit (visuomotor flexibility)</td>
<td>0.06</td>
<td>0.07</td>
<td>0.86</td>
<td>.39</td>
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<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
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<td>0.06</td>
<td>5.87</td>
<td>&lt; .001</td>
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<tr>
<td></td>
<td>~ Tracking * Hyperactive-Impulsive Sx</td>
<td>0.08</td>
<td>0.08</td>
<td>0.96</td>
<td>.34</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Pursuit * Hyperactive-Impulsive Sx</td>
<td>0.05</td>
<td>0.08</td>
<td>0.61</td>
<td>.54</td>
<td></td>
</tr>
<tr>
<td>A4</td>
<td>Dimensional Change Card Sort (cognitive flexibility)</td>
<td>0.03</td>
<td>0.00</td>
<td>0.06</td>
<td>0.01</td>
<td>.99</td>
</tr>
<tr>
<td></td>
<td>Intercept</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping (motor generation)</td>
<td>0.09</td>
<td>0.07</td>
<td>1.38</td>
<td>.17</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tracking (visuomotor fluency)</td>
<td>0.04</td>
<td>0.07</td>
<td>0.48</td>
<td>.63</td>
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<tr>
<td></td>
<td>~ Pursuit (visuomotor flexibility)</td>
<td>0.15</td>
<td>0.07</td>
<td>2.09</td>
<td>.037</td>
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<tr>
<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
<td>0.06</td>
<td>0.06</td>
<td>0.94</td>
<td>.35</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping * Hyperactive-Impulsive Sx</td>
<td>-0.01</td>
<td>0.08</td>
<td>-0.13</td>
<td>.90</td>
<td></td>
</tr>
</tbody>
</table>

NB. Bold text denotes statistically significant results. The alpha level was 0.05 for each model. Hyp: Relevant hypothesis. Sx: symptoms.

Part A: Hypotheses Regarding Associations Between Specific Aspects of Motor and Cognitive Control

Hypothesis 1: Motor generation and visuomotor fluency would be positively associated with cognitive inhibition. A multiple linear regression model containing Tapping (motor generation), Tracking (visuomotor fluency), Hyperactive-Impulsive Symptoms, and
Tapping/Tracking and Hyperactive-Impulsive Symptoms interaction terms significantly predicted 5% of the variance in Flanker (cognitive inhibition) performance (Adjusted $R^2 = 0.08$, $F(4, 250) = 4.40, p = .002$). Only Tracking was a significant predictor of Flanker (cognitive inhibition) performance within the model ($\beta = 0.21$, 95% CI = 0.09, 0.34, $p = .001$).

**Hypothesis 2: Motor generation would be positively associated with working memory.** A model containing Tapping (motor generation), Hyperactive-Impulsive Symptoms, and a Tapping and Hyperactive-Impulsive Symptoms interaction term did not significantly predict Backwards Digit Recall (verbal working memory) performance (Adjusted $R^2 = 0.01$, $F(3,251) = 1.91, p = 0.13$). However, a model containing identical terms did significantly explain 13% of the variance in Mister X (visuospatial working memory) performance (Adjusted $R^2 = 0.13$, $F(3.251) = 13.86, p < .001$); although, only Hyperactive-Impulsive Symptoms was a significant predictor within the model ($\beta = 0.38$, 95% CI = 0.26, 0.5, $p = <.001$).

**Hypothesis 3: Visuomotor fluency and visuomotor flexibility would be positively associated with working memory manipulation.** A model containing Tracking (visuomotor fluency), Pursuit (visuomotor flexibility), Hyperactive-Impulsive Symptoms and Tracking/Pursuit and Hyperactive-Impulsive Symptoms interaction terms collectively accounted for 12% of the variance in Mister X Processing (visuospatial working memory manipulation): Adjusted $R^2 = 0.12$, $F(5,249) = 8.14, p < .001$). Only Hyperactive-Impulsive Symptoms were a significant predictor within the model ($\beta = 0.36$, 95% CI = 0.26, 0.50, $p < .001$). Tracking (visuomotor fluency)/Pursuit (visuomotor flexibility) by Hyperactive-Impulsive Symptoms moderation terms were not significant predictors (see Table 4.)

**Hypothesis 4: Motor generation, visuomotor fluency, and visuomotor flexibility would be positively associated with cognitive flexibility.** A model containing Tapping
(motor generation), Tracking (visuomotor fluency), Pursuit (visuomotor flexibility), Hyperactive-Impulsive Symptoms and Tapping/Tracking/Pursuit and Hyperactive-Impulsive Symptoms interaction terms predicted 3% of the variance on the Dimensional Change Card Sort task (Adjusted $R^2 = 0.03$, $F(5,249) = 2.55$, $p = 0.029$). Only Pursuit was a significant predictor within the model ($\beta = 0.15$, 95% CI = 0.01, 0.3, $p = .037$).

**Hypothesis 5: Greater motor generation in children with higher levels of hyperactive-impulsive symptoms would be associated with poorer cognitive inhibition, working memory, and cognitive flexibility.** The Tapping (motor generation) by Hyperactive-Impulsive Symptoms moderation terms in models for all cognitive inhibition, working memory, and cognitive flexibility were all non-significant (see Table 4). Accordingly, post-hoc simple slopes analysis was not used.

**Hypothesis 6: Children with higher hyperactive-impulsive symptoms and poorer visuomotor fluency and visuomotor flexibility would display even poorer working memory manipulation.** The Tracking (visuomotor fluency) and Pursuit (visuomotor flexibility) by Hyperactive-Impulsive Symptoms moderation terms in the Mister X Processing (working memory manipulation) multiple linear regression model were both non-significant (see Table 4), meaning post-hoc simple slopes analysis was not used.

**Part B: Hypotheses Regarding Specific Processes Underlying Cognitive Inhibition**

Multiple linear regression analyses regarding our hypotheses that 1) motor generation ability would be positively associated with stimuli encoding and motor response execution time underlying cognitive inhibition, 2) visuomotor fluency would be associated with the speed-accuracy trade-off underlying cognitive inhibition, and 3) motor generation and visuomotor fluency would be positively associated with processing efficiency underlying cognitive inhibition are reported in Table 5. In summary, these analyses of
revealed that the hypothesised motor control variables did not significantly predict component processes of NIH Flanker (cognitive inhibition) performance. Correlational analysis did not reveal any significant associations amongst cognitive modelling variables and NIH Flanker performance (see Appendix K); we consider potential explanations for this in our discussion.

Table 5. Multiple Linear Regression Analyses of Motor Control and Cognitive Modelling Variables.

<table>
<thead>
<tr>
<th>Hyp.</th>
<th>Dependent and predictor variables</th>
<th>Adj. R²</th>
<th>Est.</th>
<th>SE</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1</td>
<td>Drift rate (processing efficiency)</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td>.39</td>
</tr>
<tr>
<td></td>
<td>Intercept</td>
<td>0.00</td>
<td>0.08</td>
<td>-0.04</td>
<td>.97</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping (motor generation)</td>
<td>-0.06</td>
<td>0.08</td>
<td>-0.75</td>
<td>.46</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tracking (visuomotor fluency)</td>
<td>0.16</td>
<td>0.08</td>
<td>1.88</td>
<td>.06</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
<td>-0.05</td>
<td>0.08</td>
<td>-0.55</td>
<td>.58</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping * Hyperactive-Impulsive Sx</td>
<td>-0.06</td>
<td>0.11</td>
<td>-0.55</td>
<td>.59</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tracking * Hyperactive-Impulsive Sx</td>
<td>0.09</td>
<td>0.09</td>
<td>0.96</td>
<td>.34</td>
<td></td>
</tr>
<tr>
<td>B2</td>
<td>Boundary separation (speed-accuracy trade-off)</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td>.51</td>
</tr>
<tr>
<td></td>
<td>Intercept</td>
<td>0.00</td>
<td>0.08</td>
<td>0.00</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tracking (visuomotor fluency)</td>
<td>-0.11</td>
<td>0.08</td>
<td>-1.33</td>
<td>0.18</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
<td>-0.06</td>
<td>0.08</td>
<td>-0.75</td>
<td>0.45</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tracking * Hyperactive-Impulsive Sx</td>
<td>-0.02</td>
<td>0.09</td>
<td>-0.18</td>
<td>0.86</td>
<td></td>
</tr>
<tr>
<td>B3</td>
<td>Nondecision time (stimuli encoding and motor response execution)</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
<td>.17</td>
</tr>
<tr>
<td></td>
<td>Intercept</td>
<td>-0.02</td>
<td>0.08</td>
<td>-0.19</td>
<td>.85</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping (motor generation)</td>
<td>0.11</td>
<td>0.08</td>
<td>1.33</td>
<td>.18</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Hyperactive-Impulsive Sx</td>
<td>-0.09</td>
<td>0.08</td>
<td>-1.12</td>
<td>.26</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~ Tapping * Hyperactive-Impulsive Sx</td>
<td>-0.16</td>
<td>0.10</td>
<td>-1.58</td>
<td>.12</td>
<td></td>
</tr>
</tbody>
</table>

NB. The alpha level was set at 0.05. Hyp: Hypothesis. Sx: Symptoms.
Discussion

Motor and cognitive control difficulties co-occur in childhood ADHD. Indeed, difficulties with motor control may contribute to difficulties with cognitive control across the ADHD continuum (Koziol et al., 2013). Accordingly, motor control is a candidate target for early intervention to improve cognitive outcomes. However, the relationship between motor control and cognitive control is poorly understood. We sought to clarify which specific aspects of motor and cognitive control are related in children, and to test if these relationships are moderated by hyperactive-impulsive symptoms.

Our hypothesis that children's cognitive inhibition would be associated with their motor generation and visuomotor fluency was partly supported. Our analyses revealed that performance on a test of visuomotor fluency predicted a small portion of the variance in performance on a measure of cognitive inhibition. Children with better visuomotor fluency displayed better cognitive inhibition, which is consistent with the hypothesis that cognitive inhibition relies on this motor skill, although our cross-sectional analyses are not sufficient to demonstrate a causal relationship. In contrast, no evidence was found in favour of an association between motor generation and cognitive inhibition. Together, our findings imply that the ability to visually control movement in relation to predictable visual stimuli is associated with the ability to mentally inhibit the effects of distracting information. This interpretation is broadly consistent with previous research suggesting that manual dexterity is positively associated with cognitive inhibition (Livesey et al., 2006; Rigoli et al., 2012; Stöckel & Hughes, 2016). Moreover, our findings imply that it is the visual control of motor responses (i.e., visuomotor fluency) rather than the generation of motor actions that is linked with cognitive inhibition.

Additionally, our hypothesis that children’s cognitive flexibility is associated with their motor generation, visuomotor fluency, and visuomotor flexibility abilities, was partially
supported. A test of visuomotor flexibility was significantly associated with performance on the Dimensional Change Cart Sort task, which is a measure of cognitive flexibility. By contrast, cognitive flexibility was not associated with tests of motor generation or visuomotor fluency. Our findings suggest that being able to visually control movement in response to unpredictable visual stimuli is associated with the ability to change focus from one frame of mind to another. These results are consistent with a previous study which reported that visuomotor integration and motor coordination are positively associated with cognitive flexibility measured by an adapted Dimensional Change Card Sort Task (Fang et al., 2017). Moreover, our findings indicate that it is the ability to visually control movement in unpredictable situations, but not the ability to generate consistent movements over time or visually control movement in predictable situations, that underlies the association between visuomotor control and cognitive flexibility.

Our results did not support our other hypotheses. For example, we reasoned that children’s ability to generate persistent motor output over time would support their ability to maintain information in working memory over time. However, neither verbal nor visuospatial working memory performance were associated with a test of motor generation. Additionally, visuospatial working memory manipulation was not associated with tests of visuomotor fluency and visuomotor flexibility, despite our theorising that performing predictable and spontaneous visually guided movements was akin to manipulating information held in working memory. While the absence of evidence is not the same as evidence of absence, our findings indicate that motor generation, visuomotor fluency, and visuomotor flexibility are not strongly associated with working memory. Here, our findings contrast those from previous studies which found that motor skills are weakly but positively associated with verbal working memory manipulation (Wassenberg et al., 2005) and visuospatial working memory capacity (Rigoli et al., 2012; Stöckel & Hughes, 2016). One potential reason for this
discrepancy is that working memory, like cognitive inhibition and cognitive flexibility, is a broad construct encompassing several dissociable subprocesses (Baddeley, 2012). It is possible that only certain working memory subprocesses depend on motor control and that these elements were better tapped by the measures used or the association was stronger in the samples used in previous studies.

Building on Koziol et al.’s (2013) suggestion that motor control contributes to difficulties with cognitive control across the ADHD continuum (Koziol et al., 2013), we hypothesised that hyperactive-impulsive symptoms would moderate associations between motor control and cognitive control. Specifically, we reasoned that associations between motor generation and several aspects of cognitive control (cognitive inhibition, working memory, and cognitive flexibility) are reversed in children with higher hyperactive-impulsive symptoms. We made this hypothesis considering Sergeant’s (2000, 2005) theorising around levels of physiological activation having a non-linear association with cognitive processing with both too much and too little activation undermining performance. Accordingly, we expected the relationship between motor generation and executive functioning abilities to be reversed in children with higher levels of hyperactive-impulsive symptoms because children with these traits can exhibit too much physiological activation (Burley et al., 2021; Murillo et al., 2015). We also suggested that children with lower visuomotor fluency and visuomotor flexibility abilities and higher levels of hyperactive-impulsive symptoms would display poorer levels of working memory manipulation than children with lower levels of hyperactive-impulsive symptoms. Contrary to our expectations, we did not find any evidence that associations between specific aspects of motor control and cognitive control were moderated by hyperactive-impulsive symptoms. Taken together, our results indicate that children with different levels of hyperactive-impulsive symptoms have similar relationships between specific aspects of motor control and cognitive control.
Another aim of our study was to understand the relationship between motor control and cognitive inhibition (e.g., Livesey et al., 2006; Stöckel & Hughes, 2016; Rigoli et al., 2012) in more detail. To this end, we used cognitive modelling to break down the Flanker task performance into processing efficiency, speed-accuracy trade-off, and stimuli encoding and motor response execution time components (Ratcliff & McKoon, 2008; Wagenmakers et al., 2007). We predicted that these component processes would be differentially associated with motor generation, visuomotor fluency, and visuomotor flexibility. However, in contrast with our expectations, no aspect of motor control was significantly associated with any component processes. Paradoxically, while our cognitive modelling was acceptably robust, correlational analysis suggested that none of the component processes were significantly associated with the NIH Flanker score. One explanation is that the NIH Flanker task uses a complex scoring method (see measures section for an explanation) which can include a combination of reaction time and accuracy data or just accuracy data, depending on whether a child meets an accuracy criterion (Zelazo et al., 2013). These scores are then standardised with reference to a normative sample. By contrast, our cognitive modelling methods used raw reaction time and accuracy data from the Flanker task, with reaction times exceeded three seconds being discarded. Subsequently, the NIH Flanker score and the cognitive modelling components may have been sensitive to different aspects of Flanker task performance. To aid interpretability, future studies should use traditional task scores and cognitive models based on identical data.

Our study can inform cognitive remediation interventions for children with motor and cognitive differences (e.g., Meyer et al., 2020; Pauli-Pott et al., 2021). Specifically, our findings draw attention to difficulties in visually controlling movement as a potential target for early intervention to minimise the risk of poor cognitive control outcomes, which are predictive of poorer life outcomes (Moffit et al., 2011). Treating visuomotor control issues
might improve cognitive outcomes, given that the development of motor control begins before the development of cognitive control (Njiokiktjien, 2007; Piek et al., 2008). Training on visuomotor fluency and visuomotor flexibility tasks might lead to improvement in cognitive inhibition and cognitive flexibility abilities. Improving visuomotor fluency and visuomotor flexibility might also serve as a useful adjunct to exercise-based interventions. There is systematic review evidence for exercise as an intervention to improve cognitive control in typically developing children (Bidzan-Bluma & Lipowska, 2018) and those with a diagnosis of ADHD (Den Heijer et al., 2017). Aerobic exercises (e.g., running) appear to be particularly beneficial for cognitive functioning. Emphasising visuomotor control skills (e.g., passing a baton in a relay) during this type of exercise might result in incremental cognitive benefits.

There are at least four limitations with our study. First, while our study design enabled the investigation of individual differences in hyperactive-impulsive symptoms, it did not enable consideration of clinical versus non-clinical group differences. Such comparisons would not have been appropriate given that only a small minority of children in our sample (maximum 26%) met diagnostic criteria for ADHD and fewer still met criteria for the hyperactive-impulsive subtype (6.3%) where these symptoms predominate. Still, a mixed modelling approach would facilitate the investigation of relationships amongst motor control, cognitive control, and hyperactive-impulsive symptoms in the context of whether or not children cross clinical thresholds for ADHD.

Second, there are also potential limitations with the simultaneous entry multiple linear regression approach employed. Simultaneous entry regression was used instead of hierarchical regression because of the absence of prior research looking specifically at motor generation, visuomotor fluency, and visuomotor flexibility, which meant that there was not an obvious principled way to dictate which motor variables to enter in which order as part of
a hierarchical approach. It has been argued that simultaneous entry is the most appropriate method for hypothesis testing (Studenmund & Cassidy, 1987) as opposed to exploratory research. However, simultaneous entry is less appropriate when there is a high number of candidate predictors (Kucuck et al., 2016); for example, in our tests of hypotheses three and four which both involved five predictors in total, including two interaction terms. To address this limitation, future research can use our preliminary findings associating visuomotor fluency with cognitive inhibition and visuomotor flexibility with cognitive flexibility as the basis for theoretically motivated hierarchical linear regression models.

Third, while our study establishes statistical associations between specific aspects of motor control and cognitive control, it does not demonstrate causal relationships. To investigate causality, future research could use longitudinal methods to confirm that motor control differences/difficulties precede cognitive differences/difficulties and experimental method, such as increasing the motor demands of cognitive control tasks to investigate a direct effect of motor control on cognitive control. Additionally, treatment studies based on the clinical implications of our study could provide evidence of causal relationships. For example, if an intervention targeting visual control of movement in unpredictable settings led to cognitive improvements in flexibly switching between frames of mind, this would imply that motor flexibility directs cognitive flexibility.

Finally, the generalisability of our findings associating visuomotor fluency with inhibition and visuomotor flexibility and cognitive flexibility are unclear. The Flanker task involves the inhibition of attention in the context of distracting visual stimuli and the Dimensional Change Card Sort Task involves cognitive flexibility in the context of visual stimuli. Other tests of cognitive inhibition and cognitive flexibility were not used although it is known that other tests may probe differing neurocognitive mechanisms (Kornblum, 1994; Paap et al., 2020). It remains to be seen whether the associations established in our study
generalise to other aspects of cognitive inhibition and flexibility, such as in the verbal domain (e.g., Burgess & Shallice, 1997).
Conclusion

In conclusion, our study identifies two links between specific aspects of motor control and cognitive control. First, the ability to fluently perform visually guided movement in predictable contexts is weakly associated with the ability to cognitively inhibit the effect of conflicting visual information. Second, the ability to flexibly perform visually guided movement in unpredictable contexts is weakly associated with the ability to flexibly shift attention from one frame of mind to another. Contrary to our hypotheses, these relationships appear to be quantitatively (i.e., of a similar strength) and qualitatively (i.e., of the same direction) similar across the hyperactive-impulsive continuum in childhood. That is, children with low and high levels of hyperactive-impulsive symptoms display similar relationships between motor and cognitive control. Unfortunately, only a small proportion of adolescents in the sample met clinical criteria for ADHD and causal statements about the influence of motor control on cognitive control cannot be made as the study was cross-sectional. In addition to helping to clarify the theoretically important but poorly understood relationships between motor and cognitive control, our findings indicate that early interventions and adjunctive treatments targeting visuomotor control might incrementally benefit cognitive functioning in childhood. Moreover, our findings suggest that such interventions might be equally beneficial for children with high and low levels of hyperactive-impulsive symptoms.
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https://doi.org/10.1037/neu0000636


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https://doi.org/10.1111/j.1467-8624.2005.00899.x


Appendix A: PROSPERO Registration for Paper 1

PROSPERO
International prospective register of systematic reviews

University of York
Centre for Reviews and Dissemination

Systematic review

This record cannot be edited because it has been marked as out of scope

1. Title
   Give the title of the review in English
   The relationship between executive functioning and depressive symptoms in adolescence: a systematic review and meta-analysis

2. Original language title.
   For reviews in languages other than English, give the title in the original language. This will be displayed with the English language title.

3. *Anticipated or actual start date.
   Give the date the systematic review started or is expected to start.
   29/11/2021

4. *Anticipated completion date.
   Give the date by which the review is expected to be completed.
   28/02/2022

5. *Stage of review at time of this submission.
   This field uses answers to initial screening questions. It cannot be edited until after registration.
   Tick the boxes to show which review tasks have been started and which have been completed.
   Update this field each time any amendments are made to a published record.

The review has not yet started: No
**PROSPERO**  
**International prospective register of systematic reviews**

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<th>Started</th>
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<td>No</td>
</tr>
<tr>
<td>Piloting of the study selection process</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Formal screening of search results against eligibility criteria</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Data extraction</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Risk of bias (quality) assessment</td>
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<td>No</td>
</tr>
<tr>
<td>Data analysis</td>
<td>No</td>
<td>No</td>
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</table>

Provide any other relevant information about the stage of the review here.

6. **Named contact.**  
The named contact is the guarantor for the accuracy of the information in the register record. This may be any member of the review team.

Cameron Ferguson  
Email salutation (e.g. "Dr Smith" or "Joanne") for correspondence:

Mr Ferguson

7. **Named contact email.**  
Give the electronic email address of the named contact.

fergusonc3@cardiff.ac.uk

8. **Named contact address**

Give the full institutional/organisational postal address for the named contact.

South Wales Doctoral Programme in Clinical Psychology, School Of Psychology, Cardiff University, Floor 11,  
Tower Building, 63 Park Pl, Cardiff CF10 3AS

9. **Named contact phone number.**  
Give the telephone number for the named contact, including international dialling code.

07572021817

10. **Organisational affiliation of the review.**  
Full title of the organisational affiliations for this review and website address if available. This field may be completed as 'None' if the review is not affiliated to any organisation.

Cardiff University  
Organisation web address:
11. *Review team members and their organisational affiliations.*

Give the personal details and the organisational affiliations of each member of the review team. Affiliation refers to groups or organisations to which review team members belong. NOTE: email and country now MUST be entered for each person, unless you are amending a published record.

Mr Cameron Ferguson. Cardiff University
Dr Chris Hotson. Cardiff University
Dr Cerith Waters. Cardiff University

12. *Funding sources/sponsors.*

Details of the individuals, organizations, groups, companies or other legal entities who have funded or sponsored the review.

Supported by the South Wales Doctoral Programme in Clinical Psychology as part of Cameron Ferguson’s training as a Clinical Psychologist

Grant number(s)
State the funder, grant or award number and the date of award

13. *Conflicts of interest.*

List actual or perceived conflicts of interest (financial or academic).

None

14. **Collaborators.**

Give the name and affiliation of any individuals or organisations who are working on the review but who are not listed as review team members. NOTE: email and country must be completed for each person, unless you are amending a published record.

15. **Review question.**

State the review question(s) clearly and precisely. It may be appropriate to break very broad questions down into a series of related more specific questions. Questions may be framed or refined using P(I)E(C)OS or similar where relevant.

Are depression symptoms and executive functioning related in adolescence? Which executive functions, if any, are related to depressive symptoms?

16. **Searches.**

State the sources that will be searched (e.g. Medline). Give the search dates, and any restrictions (e.g. language or publication date). Do NOT enter the full search strategy (it may be provided as a link or attachment below.)

PubMed. Web of Science. PsycINFO. There are no date restrictions but only records in the English language will be considered. Non-peer reviewed records will not be considered.

12. **Search strategy.**

Upload a file with your search strategy, or an example of a search strategy for a specific database, (including the keywords) in pdf or word format. In doing so you are consenting to the file being made publicly accessible. Or provide a URL or link to the strategy. Do NOT provide links to your search results.
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PROSPERO
International prospective register of systematic reviews

https://www.crd.york.ac.uk/PROSPEROFILES/293983_STRATEGY_20220220.pdf

Alternatively, upload your search strategy to CRD in pdf format. Please note that by doing so you are consenting to the file being made publicly accessible.

Do not make this file publicly available until the review is complete.

18. **change** or domain being studied.
Give a short description of the disease, condition or healthcare domain being studied in your systematic review.

**Depressive symptoms**

Specify the participants or populations being studied in the review. The preferred format includes details of both inclusion and exclusion criteria.

Adolescents (aged 11-18) who may display clinical levels of anxiety and depression without bipolar, psychosis, neurodevelopmental disorders, neurological disorders, or medical illnesses known to affect executive functioning.

20. * Intervention(s), exposure(s).
Give full and clear descriptions or definitions of the interventions or the exposures to be reviewed. The preferred format includes details of both inclusion and exclusion criteria.

Not applicable.

21. * Comparator(s)/control.
Where relevant, give details of the alternatives against which the intervention/exposure will be compared (e.g. another intervention or a non-exposed control group). The preferred format includes details of both inclusion and exclusion criteria.

Not applicable.

22. **types** of study to be included.
Give details of the study designs (e.g. RCT) that are eligible for inclusion in the review. The preferred format includes both inclusion and exclusion criteria. If there are no restrictions on the types of study, this should be stated.

Cross sectional studies. Cohort studies if depressive symptoms and executive functioning were assessed concurrently.

23. **Measures**.
Give summary details of the setting or other relevant characteristics, which help define the inclusion or exclusion criteria.

**Assessment of executive functioning and depressive symptoms**

Objective measure of at least one aspect of executive functioning

Psychometric measurement of depressive symptoms

Mean age between 11 and 18 years
Zero order correlations
Single regressions

Exclusion:
Neurodevelopmental disorder and no healthy control group
Neurological disorder no healthy control group
Medical disorder known to affect executive functioning in children (e.g., diabetes) and no healthy control group
Mean age too young or too old (i.e., lower than 11 years or higher than 18 years)
Non-zero order correlation or single regression analysis (e.g., partial correlations, multiple regressions, confirmatory factor analysis, network analysis)

24. Change outcome(s).
Give the pre-specified main (most important) outcomes of the review, including details of how the outcome is defined and measured and when these measurement are made, if these are part of the review inclusion criteria.
Depressive symptoms.

Measures of effect
Please specify the effect measure(s) for your main outcome(s) e.g. relative risks, odds ratios, risk difference, and/or number needed to treat.
Correlations or simple regressions from which correlations can be derived.

25. * Additional outcome(s).
List the pre-specified additional outcomes of the review, with a similar level of detail to that required for main outcomes. Where there are no additional outcomes please state ‘None’ or ‘Not applicable’ as appropriate to the review
Not applicable.

Measures of effect
Please specify the effect measure(s) for your additional outcome(s) e.g. relative risks, odds ratios, risk difference, and/or number needed to treat.

26. Change extraction (selection and coding).
Describe how studies will be selected for inclusion. State what data will be extracted or obtained. State how this will be done and recorded.
Definements assessment of executive functioning and depressive symptoms
Objective measure of at least one aspect of executive functioning
Psychometric measurement of depressive symptoms symptoms
Mean age between 11 and 18 years
Zero order correlations
Single regressions
Exclusion:

- Neurodevelopmental disorder and no healthy control group
- Neurological disorder no healthy control group
- Medical disorder known to affect executive functioning in children (e.g., diabetes) and no healthy control group
- Mean age too young or too old (i.e., lower than 11 years or higher than 18 years)
- Non zero order correlation or single regression analysis (e.g., partial correlations, multiple regressions, confirmatory factor analysis, network analysis)

Correlation coefficients and p values will be extracted and recorded with the Covidence platform. Single regression analyses will be translated into correlation coefficients and extracted as above.

**47. Bias (quality) assessment.**

State which characteristics of the studies will be assessed and/or any formal risk of bias/quality assessment tools that will be used.

The Quality Assessment Tools for Studies with Diverse Designs (QATSDD) will be used at study level to assess study quality regarding an explicit theoretical framework, sample suitability, measure suitability, design suitability, analysis suitability, and service user involvement. All eligible studies will be rated by one reviewer and 50% will also be rated by another reviewer who is blind to the first reviewer's judgements. Both reviewers will then discuss results and any discrepancies will be resolved by reconsidering the QATSDD criteria together to reach a consensus. The results of the quality assessment will be discussed in the paper but not dictate inclusion in synthesis and meta-analysis, given that medium to low quality studies are expected in the field. Conclusions will be more tentative in the case of generally poor quality across studies.

**48. Strategy for data synthesis.**

Describe the methods you plan to use to synthesise data. This must not be generic text but should be specific to your review and describe how the proposed approach will be applied to your data. If meta-analysis is planned, describe the models to be used, methods to explore statistical heterogeneity, and software package to be used.

Meta-analyses of correlation coefficients with random effects will be performed in R studio. The correlation coefficients will be between measures of internalising or depression symptoms and measures of an executive function (e.g., attention shifting). The minimum number of studies per meta-analysis (denoted in section 29) is 3. The correlation coefficients will be combined using the DerSimonian-Laird method. Single regression coefficients will be converted into correlations beforehand.

**49. Analysis of subgroups or subsets.**

State any planned investigation of 'subgroups'. Be clear and specific about which type of study or participant will be included in each group or covariate investigated. State the planned analytic approach.
Within the systematic review and meta-analysis, the following meta-analyses are planned, assuming there is a sufficient number of trials:
- depressive symptoms and interference control of distraction
- depressive symptoms and interference control of automatic responses
- depressive symptoms and working memory
- depressive symptoms and “hot” executive functioning

30. Type and method of review.
Select the type of review, review method and health area from the lists below.

Type of review
Cost effectiveness
No
Diagnostic
No
Epidemiologic
No
Individual patient data (IPD) meta-analysis
No
Intervention
No
Living systematic review
No
Meta-analysis
Yes
Methodology
No
Narrative synthesis
No
Network meta-analysis
No
Pre-clinical
No
Prevention
No
Prognostic
No
Prospective meta-analysis (PMA)
No
Review of reviews
No
Service delivery
No
Synthesis of qualitative studies
No
Systematic review
Yes
Other
No

Health area of the review
Alcohol/substance misuse/abuse
No
Blood and immune system
No
Cancer
No
Cardiovascular
No
Care of the elderly
No
Child health
Yes
Complementary therapies
No
COVID-19
No
Crime and justice
No
Dental
No
Digestive system
No
Ear, nose and throat
No
Education
No
Endocrine and metabolic disorders
No
Eye disorders
No
PROSPERO
International prospective register of systematic reviews

<table>
<thead>
<tr>
<th>Category</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>General interest</td>
<td>No</td>
</tr>
<tr>
<td>Genetics</td>
<td>No</td>
</tr>
<tr>
<td>Health inequalities/health equity</td>
<td>No</td>
</tr>
<tr>
<td>Infections and infestations</td>
<td>No</td>
</tr>
<tr>
<td>International development</td>
<td>No</td>
</tr>
<tr>
<td>Mental health and behavioural conditions</td>
<td>Yes</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>No</td>
</tr>
<tr>
<td>Neurological</td>
<td>No</td>
</tr>
<tr>
<td>Nursing</td>
<td>No</td>
</tr>
<tr>
<td>Obstetrics and gynaecology</td>
<td>No</td>
</tr>
<tr>
<td>Oral health</td>
<td>No</td>
</tr>
<tr>
<td>Palliative care</td>
<td>No</td>
</tr>
<tr>
<td>Perioperative care</td>
<td>No</td>
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<td>Physiotherapy</td>
<td>No</td>
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<tr>
<td>Pregnancy and childbirth</td>
<td>No</td>
</tr>
<tr>
<td>Public health (including social determinants of health)</td>
<td>No</td>
</tr>
<tr>
<td>Rehabilitation</td>
<td>No</td>
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<tr>
<td>Respiratory disorders</td>
<td>No</td>
</tr>
<tr>
<td>Service delivery</td>
<td>No</td>
</tr>
<tr>
<td>Skin disorders</td>
<td>No</td>
</tr>
<tr>
<td>Social care</td>
<td>No</td>
</tr>
<tr>
<td>Surgery</td>
<td>No</td>
</tr>
</tbody>
</table>
Tropical Medicine
No

Urological
No

Wounds, injuries and accidents
No

Violence and abuse
No

31. Language.
Select each language individually to add it to the list below, use the bin icon to remove any added in error.
English
There is not an English language summary

Select the country in which the review is being carried out. For multi-national collaborations select all the countries involved.
Wales

33. Other registration details.
Name any other organisation where the systematic review title or protocol is registered (e.g. Campbell, or The Joanna Briggs Institute) together with any unique identification number assigned by them. If extracted data will be stored and made available through a repository such as the Systematic Review Data Repository (SRDR), details and a link should be included here. If none, leave blank.

34. Reference and/or URL for published protocol.
If the protocol for this review is published provide details (authors, title and journal details, preferably in Vancouver format)
Add web link to the published protocol.
Or, upload your published protocol here in pdf format. Note that the upload will be publicly accessible.
No I do not make this file publicly available until the review is complete
Please note that the information required in the PROSPERO registration form must be completed in full even if access to a protocol is given.

35. Dissemination plans.
Do you intend to publish the review on completion?

Yes
Give brief details of plans for communicating review findings?
To be submitted to peer reviewed journal.

36. Keywords.
PROSPERO
International prospective register of systematic reviews

Give words or phrases that best describe the review. Separate keywords with a semicolon or new line. Keywords help PROSPERO users find your review (keywords do not appear in the public record but are included in searches). Be as specific and precise as possible. Avoid acronyms and abbreviations unless these are in wide use.

Depression
Executive functioning
Cognitive control

37. Details of any existing review of the same topic by the same authors.
If you are registering an update of an existing review give details of the earlier versions and include a full bibliographic reference, if available.

38. * Current review status.
Update review status when the review is completed and when it is published. New registrations must be ongoing so this field is not editable for initial submission.
Please provide anticipated publication date

39. Any additional information.
Provide any other information relevant to the registration of this review.

40. [ ] Name of final report/publication(s) or preprints if available.
Leave empty until publication details are available OR you have a link to a preprint (NOTE: this field is not editable for initial submission). List authors, title and journal details preferably in Vancouver format.
Give the link to the published review or preprint.
# Appendix B: Application of the Quality Assessment Tool for Studies with Diverse Designs in Paper 1

| Study                          | Q1 | Q2 | Q3 | Q4 | Q5 | Q6 | Q7 | Q8 | Q9 | Q10 | Q11 | Q12 | Q13 | Q14 | Q15 | Q16 | Sum | %   | R2 Sum | %   | Av. Sum | %   | Av. Sum | %   |
|-------------------------------|----|----|----|----|----|----|----|----|----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-------|-----|---------|-----|---------|-----|---------|-----|
| Davidovich et al. (2016)      | 1  | 3  | 2  | 0  | 2  | 2  | 1  | 2  | 0  | 3   | NA  | 3  | 2  | NA  | 0  | 2  | 24  | 57.1 | 21  | 50.0  | 22.5 | 53.6    |
| Dickson et al. (2017)         | 3  | 3  | 1  | 0  | 2  | 3  | 2  | 3  | 3  | 2   | NA  | 2  | 0  | NA  | 0  | 1  | 25  | 59.5 |      |        |        |        |
| Evans et al. (2016)           | 3  | 3  | 0  | 0  | 2  | 2  | 1  | 1  | 3  | 2   | NA  | 3  | 2  | NA  | 0  | 3  | 25  | 59.5 |      |        |        |        |
| Gray et al. (2016)            | 3  | 3  | 3  | 1  | 2  | 2  | 1  | 2  | 1  | 2   | NA  | 3  | 2  | NA  | 0  | 2  | 27  | 64.3 | 28  | 66.7  | 27.5 | 65.5    |
| Han et al. (2016)             | 1  | 3  | 1  | 0  | 2  | 0  | 2  | 2  | 0  | 3   | NA  | 3  | 0  | NA  | 0  | 2  | 19  | 45.2 |      |        |        |        |
| Han et al. (2012)             | 2  | 3  | 3  | 0  | 2  | 2  | 2  | 2  | 0  | 2   | NA  | 3  | 0  | NA  | 0  | 1  | 22  | 52.4 |      |        |        |        |
| Jandrić et al. (2021)         | 2  | 2  | 2  | 0  | 2  | 2  | 2  | 2  | 3  | 3   | NA  | 2  | 0  | NA  | 0  | 2  | 24  | 57.1 |      |        |        |        |
| Kavannaugh et al. (2012)      | 2  | 2  | 3  | 0  | 2  | 1  | 2  | 1  | 0  | 2   | NA  | 2  | 0  | NA  | 0  | 1  | 18  | 42.9 |      |        |        |        |
| Kim et al. (2021)             | 2  | 3  | 3  | 1  | 2  | 2  | 2  | 3  | 2   | NA  | 2  | 2  | NA  | 0  | 1  | 27  | 64.4 | 30  | 71.4  | 28.5 | 67.9    |
| Morea & Calvete (2021)        | 3  | 3  | 0  | 0  | 2  | 2  | 2  | 2  | 3  | 3   | NA  | 3  | 2  | NA  | 0  | 3  | 28  | 66.7 | 31  | 73.8  | 29.5 | 70.2    |
| Moreno-Manso et al. (2020)    | 2  | 3  | 3  | 2  | 3  | 2  | 2  | 1  | 3  | 3   | NA  | 2  | 2  | NA  | 0  | 2  | 30  | 71.4 |      |        |        |        |
| Murphy et al. (2018)          | 2  | 3  | 2  | 3  | 1  | 3  | 2  | 1  | 0  | 3   | NA  | 2  | 2  | NA  | 0  | 3  | 27  | 64.3 |      |        |        |        |
| Peters et al. (2019)          | 2  | 3  | 1  | 3  | 1  | 2  | 1  | 2  | 3  | 3   | NA  | 2  | 0  | NA  | 0  | 2  | 25  | 59.5 |      |        |        |        |
| Rifkin et al. (2021)          | 2  | 3  | 2  | 2  | 2  | 2  | 2  | 2  | 3  | 3   | NA  | 3  | 2  | NA  | 0  | 2  | 30  | 71.4 |      |        |        |        |
| Sommerfeldt et al. (2015)     | 3  | 3  | 1  | 1  | 2  | 2  | 1  | 1  | 0  | 2   | NA  | 2  | 1  | NA  | 0  | 3  | 22  | 52.4 |      |        |        |        |
| Stewart et al. (2018)         | 3  | 3  | 3  | 2  | 3  | 3  | 1  | 2  | 2   | NA  | 2  | 2  | NA  | 0  | 3  | 32  | 76.2 |      |        |        |        |
| Valentinio et al. (2012)      | 3  | 3  | 3  | 0  | 2  | 1  | 3  | 2  | 3  | 3   | NA  | 3  | 1  | NA  | 0  | 2  | 29  | 69.1 |      |        |        |        |
| Vergara-Lopez et al. (2013)   | 3  | 3  | 2  | 0  | 1  | 1  | 2  | 1  | 1  | 2   | NA  | 3  | 2  | NA  | 0  | 2  | 23  | 54.8 | 27  | 64.3  | 25  | 59.5    |

## Quality appraisal

### Intraclass Correlation Coefficient

#### Input QATSDD ratings data

```r
QATSDD_data <- data.frame(
  R1 = c(24, 27, 27, 28, 23),  # Reviewer 1's scores
  R2 = c(21, 28, 30, 31, 27)  # Reviewer 2's scores
)
```

#### Calculate intraclass correlation coefficient

```r
ICC(QATSDD_data)
```

**Output:**

```
## Call: ICC(x = QATSDD_data)

## Intraclass correlation coefficients

<table>
<thead>
<tr>
<th>type</th>
<th>ICC</th>
<th>F</th>
<th>df1</th>
<th>df2</th>
<th>p</th>
<th>lower bound</th>
<th>upper bound</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single_raters_absolute</td>
<td>ICC1</td>
<td>0.57</td>
<td>3.7</td>
<td>4</td>
<td>5</td>
<td>0.094</td>
<td>0.17</td>
</tr>
<tr>
<td>Single_random_raters</td>
<td>ICC2</td>
<td>0.58</td>
<td>4.1</td>
<td>4</td>
<td>4</td>
<td>0.099</td>
<td>0.21</td>
</tr>
<tr>
<td>Single_fixed_raters</td>
<td>ICC3</td>
<td>0.61</td>
<td>4.1</td>
<td>4</td>
<td>4</td>
<td>0.099</td>
<td>0.33</td>
</tr>
<tr>
<td>Average_raters_absolute</td>
<td>ICC1k</td>
<td>0.73</td>
<td>3.7</td>
<td>4</td>
<td>5</td>
<td>0.094</td>
<td>0.42</td>
</tr>
<tr>
<td>Average_random_raters</td>
<td>ICC2k</td>
<td>0.73</td>
<td>4.1</td>
<td>4</td>
<td>4</td>
<td>0.099</td>
<td>0.22</td>
</tr>
<tr>
<td>Average_fixed_raters</td>
<td>ICC3k</td>
<td>0.76</td>
<td>4.1</td>
<td>4</td>
<td>4</td>
<td>0.099</td>
<td>0.55</td>
</tr>
</tbody>
</table>
Appendix D: Extracted Data and Meta-Mar Output for Meta-Analyses in Paper 1

Perseverative Errors (Cognitive Flexibility) and Depressive Symptoms

**Extracted Data**

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>r</th>
<th>subgroup</th>
<th>yi</th>
<th>vi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dickson et al. (2017)</td>
<td>86</td>
<td>0.480</td>
<td>subgroup1</td>
<td>0.480</td>
<td>0.006968049</td>
</tr>
<tr>
<td>Evans et al. (2015)</td>
<td>192</td>
<td>0.120</td>
<td>subgroup1</td>
<td>0.120</td>
<td>0.005085902</td>
</tr>
<tr>
<td>Han et al. (2016)</td>
<td>220</td>
<td>0.060</td>
<td>subgroup1</td>
<td>0.060</td>
<td>0.004533393</td>
</tr>
<tr>
<td>Kavanaugh et al. (2012)</td>
<td>105</td>
<td>-0.053</td>
<td>subgroup1</td>
<td>-0.053</td>
<td>0.009561441</td>
</tr>
<tr>
<td>Murphy et al. (2018)</td>
<td>106</td>
<td>0.090</td>
<td>subgroup1</td>
<td>0.090</td>
<td>0.009370149</td>
</tr>
<tr>
<td>Valentino et al. (2012)</td>
<td>49</td>
<td>0.380</td>
<td>subgroup1</td>
<td>0.380</td>
<td>0.015251070</td>
</tr>
<tr>
<td>Vergara-Lopez et al. (2013)</td>
<td>373</td>
<td>0.000</td>
<td>subgroup1</td>
<td>0.000</td>
<td>0.002688172</td>
</tr>
</tbody>
</table>

**Meta-Mar Output**

Number of studies combined: \( k = 7 \)
Number of observations: \( o = 1131 \)

<table>
<thead>
<tr>
<th>COR</th>
<th>95%-CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common effect model</td>
<td>0.0912 [0.0326; 0.1492]</td>
<td>3.05</td>
<td>0.0023</td>
</tr>
<tr>
<td>Random effects model</td>
<td>0.1443 [-0.0020; 0.2845]</td>
<td>1.93</td>
<td>0.0532</td>
</tr>
</tbody>
</table>

Quantifying heterogeneity:
\( \tau^2 = 0.0309 \ [0.0075; 0.2113]\); \( \tau = 0.1758 \ [0.0866; 0.4597]\)
\( I^2 = 76.4\% \ [50.5\%; 88.8\%]\); \( H = 2.06 \ [1.42; 2.98]\)

Test of heterogeneity:
\( Q \) d.f. p-value
\( 25.44 \ 6 \ 0.0003 \)

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for tau^2
- Q-profile method for confidence interval of tau^2 and tau
- Fisher's z transformation of correlations

Number of studies combined: k = 7
Number of observations: o = 1131

<table>
<thead>
<tr>
<th>ZCOR</th>
<th>95%CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common effect model</td>
<td>0.0915 [0.0326; 0.1503]</td>
<td>3.05</td>
<td>0.0023</td>
</tr>
<tr>
<td>Random effects model</td>
<td>0.1453 [-0.0020; 0.2926]</td>
<td>1.93</td>
<td>0.0532</td>
</tr>
</tbody>
</table>

Quantifying heterogeneity:
\[ \tau^2 = 0.0309 \ [0.0075; 0.2113] \]; \[ \tau = 0.1758 \ [0.0866; 0.4597] \]
\[ I^2 = 76.4\% \ [50.5\%; 88.8\%] \]; \[ H = 2.06 \ [1.42; 2.98] \]

Test of heterogeneity:
\[ Q \ d.f. \ p-value \]
\[ 25.44 \ 6 \ 0.0003 \]

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for tau^2
- Q-profile method for confidence interval of tau^2 and tau
- Fisher's z transformation of correlations

Fail-safe N Calculation Using the Rosenthal Approach

Observed Significance Level: <.0001
Target Significance Level: 0.05

Fail-safe N: 45
Cognitive Inhibition of Distraction and Depressive Symptoms

Extracted Data

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>r</th>
<th>studygroup</th>
<th>yi</th>
<th>vi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gray et al. (2016) maltreated</td>
<td>27</td>
<td>0.029</td>
<td>subgroup1</td>
<td>0.029</td>
<td>0.038396873</td>
</tr>
<tr>
<td>Gray et al. (2016) non-maltreated</td>
<td>24</td>
<td>0.129</td>
<td>subgroup1</td>
<td>0.129</td>
<td>0.042043258</td>
</tr>
<tr>
<td>Sommerfeldt et al. (2015)</td>
<td>162</td>
<td>0.250</td>
<td>subgroup1</td>
<td>0.250</td>
<td>0.005459045</td>
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</tbody>
</table>

Meta-Mar Output

Number of studies combined: k = 3
Number of observations: o = 213

<table>
<thead>
<tr>
<th></th>
<th>COR</th>
<th>95%-CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common effect model</td>
<td>0.2125</td>
<td>[0.0785; 0.3391]</td>
<td>3.08</td>
<td>0.0021</td>
</tr>
<tr>
<td>Random effects model</td>
<td>0.2125</td>
<td>[0.0785; 0.3391]</td>
<td>3.08</td>
<td>0.0021</td>
</tr>
</tbody>
</table>

Quantifying heterogeneity:

\[ \tau^2 = 0 \ [0.0000; 0.4856]; \tau = 0 \ [0.0000; 0.6969] \]
\[ I^2 = 0.0\% \ [0.0\%; 89.6\%]; H = 1.00 \ [1.00; 3.10] \]

Test of heterogeneity:

<table>
<thead>
<tr>
<th>Q</th>
<th>d.f.</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.24</td>
<td>2</td>
<td>0.5373</td>
</tr>
</tbody>
</table>

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for \( \tau^2 \)
- Q-profile method for confidence interval of \( \tau^2 \) and \( \tau \)
- Fisher's z transformation of correlations
Number of studies combined: k = 3
Number of observations: o = 213

<table>
<thead>
<tr>
<th>ZCOR</th>
<th>95%-CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
</table>
Common effect model  0.2158  [0.0786; 0.3531]  3.08  0.0021
Random effects model  0.2158  [0.0786; 0.3531]  3.08  0.0021

Quantifying heterogeneity:
tau^2 = 0  [0.0000; 0.4856];  tau = 0  [0.0000; 0.6969]
I^2 = 0.0%  [0.0%; 89.6%];  H = 1.00  [1.00; 3.10]

Test of heterogeneity:
   Q  d.f.  p-value
   1.24  2  0.5373

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for tau^2
- Q-profile method for confidence interval of tau^2 and tau
- Fisher's z transformation of correlations

Fail-safe N Calculation Using the Rosenthal Approach

Observed Significance Level: 0.0081
Target Significance Level:   0.05

Fail-safe N: 4
Cognitive Inhibition of Overlearned Responses and Depressive Symptoms

### Extracted Data

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>r</th>
<th>studygroup</th>
<th>yi</th>
<th>vi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kavannaugh et al. (2012)</td>
<td>105</td>
<td>0.125</td>
<td>subgroup1</td>
<td>0.125</td>
<td>0.009317251</td>
</tr>
<tr>
<td>Kim et al. (2021)</td>
<td>144</td>
<td>0.120</td>
<td>subgroup1</td>
<td>0.120</td>
<td>0.006793058</td>
</tr>
<tr>
<td>Moreno-Manso et al. (2021)</td>
<td>61</td>
<td>-0.084</td>
<td>subgroup1</td>
<td>-0.084</td>
<td>0.016432296</td>
</tr>
<tr>
<td>Valentino et al. (2012)</td>
<td>49</td>
<td>0.100</td>
<td>subgroup1</td>
<td>0.100</td>
<td>0.020418750</td>
</tr>
</tbody>
</table>

### Meta-Mar Output

Number of studies combined: $k = 4$
Number of observations: $o = 359$

<table>
<thead>
<tr>
<th>COR</th>
<th>95%–CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common effect model</td>
<td>0.0850 [−0.0201; 0.1881]</td>
<td>1.59</td>
<td>0.1127</td>
</tr>
<tr>
<td>Random effects model</td>
<td>0.0850 [−0.0201; 0.1881]</td>
<td>1.59</td>
<td>0.1127</td>
</tr>
</tbody>
</table>

Quantifying heterogeneity:
- $\tau^2 = 0 [0.0000; 0.1248]$; $\tau = 0 [0.0000; 0.3533]$
- $I^2 = 0.0% [0.0%; 84.7%]$; $H = 1.00 [1.00; 2.56]$

Test of heterogeneity:
- $Q$ d.f. p-value
  - 2.02 3 0.5686

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for $\tau^2$
- Q-profile method for confidence interval of $\tau^2$ and $\tau$
- Fisher's z transformation of correlations

Number of studies combined: $k = 4$
Number of observations: $o = 359$
<table>
<thead>
<tr>
<th>ZCOR</th>
<th>95%-CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common effect model 0.0852 [-0.0201; 0.1904] 1.59 0.1127</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random effects model 0.0852 [-0.0201; 0.1904] 1.59 0.1127</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Quantifying heterogeneity:

\[ \tau^2 = 0 \ [0.0000; 0.1248]; \tau = 0 \ [0.0000; 0.3533] \]

\[ I^2 = 0.0\% \ [0.0\%; 84.7\%]; H = 1.00 \ [1.00; 2.56] \]

Test of heterogeneity:

<table>
<thead>
<tr>
<th>Q</th>
<th>d.f.</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.02</td>
<td>3</td>
<td>0.5686</td>
</tr>
</tbody>
</table>

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for \( \tau^2 \)
- Q-profile method for confidence interval of \( \tau^2 \) and \( \tau \)
- Fisher's z transformation of correlations

Fail-safe N Calculation Using the Rosenthal Approach

Observed Significance Level: 0.0811
Target Significance Level: 0.05

Fail-safe N: 0
Auditory-Verbal Working Memory and Depressive Symptoms
Extracted Data

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>r</th>
<th>subgroup</th>
<th>yi</th>
<th>vi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evans et al. (2015)</td>
<td>192</td>
<td>0.150</td>
<td>subgroup1</td>
<td>0.150</td>
<td>0.005002651</td>
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<tr>
<td>Kavanaugh et al. (2012)</td>
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<td>Subgroup1</td>
<td>0.006</td>
<td>0.009614692</td>
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<td>Wagner et al. (2015)</td>
<td>486</td>
<td>0.070</td>
<td>Subgroup1</td>
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<td>0.002041699</td>
</tr>
</tbody>
</table>

Meta-Mar Output

Number of studies combined: k = 3
Number of observations: o = 783

<table>
<thead>
<tr>
<th></th>
<th>COR</th>
<th>95%-CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common effect model</td>
<td>0.0813 [0.0110; 0.1507]</td>
<td>2.27</td>
<td>0.0234</td>
<td></td>
</tr>
<tr>
<td>Random effects model</td>
<td>0.0813 [0.0110; 0.1507]</td>
<td>2.27</td>
<td>0.0234</td>
<td></td>
</tr>
</tbody>
</table>

Quantifying heterogeneity:
- tau^2 = 0 [0.0000; 0.2016]; tau = 0 [0.0000; 0.4490]
- I^2 = 0.0% [0.0%; 89.6%]; H = 1.00 [1.00; 3.10]

Test of heterogeneity:
- Q d.f. p-value
  - 1.56 2 0.4583

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for tau^2
- Q-profile method for confidence interval of tau^2 and tau
- Fisher's z transformation of correlations

Number of studies combined: k = 3
Number of observations: o = 783

<table>
<thead>
<tr>
<th></th>
<th>ZCOR</th>
<th>95%-CI</th>
<th>z</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common effect model</td>
<td>0.0815 [0.0110; 0.1519]</td>
<td>2.27</td>
<td>0.0234</td>
<td></td>
</tr>
</tbody>
</table>
Random effects model 0.0815 [0.0110; 0.1519] 2.27 0.0234

Quantifying heterogeneity:
\[ \tau^2 = 0 [0.0000; 0.2016]; \tau = 0 [0.0000; 0.4490] \]
\[ I^2 = 0.0\% [0.0\%; 89.6\%]; H = 1.00 [1.00; 3.10] \]

Test of heterogeneity:
\[ Q \quad d.f. \quad p-value \]
\[ 1.56 \quad 2 \quad 0.4583 \]

Details on meta-analytical method:
- Inverse variance method
- Restricted maximum-likelihood estimator for \( \tau^2 \)
- Q-profile method for confidence interval of \( \tau^2 \) and \( \tau \)
- Fisher's z transformation of correlations

Fail-safe N Calculation Using the Rosenthal Approach

Observed Significance Level: 0.0156
Target Significance Level: 0.05

Fail-safe N: 3
Appendix E: ‘Child and Adolescent Mental Health’ Journal Submission Guidelines for Authors for Paper 1

Author Guidelines

Why submit to Child and Adolescent Mental Health?

- An international journal with a growing reputation for publishing work of clinical relevance to multidisciplinary practitioners in child and adolescent mental health
- Ranked in ISI: 67/129 (Pediatrics); 121/156 (Psychiatry); 100/143 (Psychiatry (Social Science)); 89/131 (Psychology, Clinical).
- 7,319 institutions with access to current content, and a further 6,696 institutions in the developing world
- High international readership - accessed by institutions globally, including North America (34%), Europe (34%) and Asia-Pacific (11%)
- Excellent service provided by editorial and production offices
- Opportunities to communicate your research directly to practitioners
- Every manuscript is assigned to one of the Joint Editors as decision-making editor; rejection rate is around 82%
- Acceptance to Early View publication averages 5 weeks
- Simple and efficient online submission – visit http://mc.manuscriptcentral.com/camh_journal
- Early View – articles appear online before the paper version is published. Click here to see the articles currently available
- Authors receive access to their article once published as well as a 25% discount on virtually all Wiley books
- All articles published in CAMH are eligible for Panel A: Psychology, Psychiatry and Neuroscience in the Research Excellence Framework (REF)

1. Contributions from any discipline that further clinical knowledge of the mental life and behaviour of children are welcomed. Papers need to clearly draw out the clinical implications for mental health practitioners. Papers are published in English. As an international journal, submissions are welcomed from any country. Contributions should be of a standard that merits presentation before an international readership. Papers may assume any of the following forms: Original Articles; Review Articles; Innovations in Practice; Narrative Matters; Debate Articles.

CAMH considers the fact that services are looking at treating young adults up until the age of 25, with the evidence that brains continue to develop until the age of 25, as well as the fact that a lot of issues that affect young adults and students are also relevant and topical to older adolescents. CAMH offers a discretionary approach and will take into consideration papers that extend into young adulthood, if they are pertinent developmentally to the younger population and contribute further to a developmental perspective across adolescence and early adult years.

Authors are asked to remember that CAMH is an international journal and therefore clarification should be provided for any references that are made in submitted papers to the practice within the authors' own country. This is to ensure that the meaning is clearly
understandable for our diverse readership. Authors should make their papers as broadly applicable as possible for a global audience.

**Original Articles:** Original Articles make an original contribution to empirical knowledge, to the theoretical understanding of the subject, or to the development of clinical research and practice.

**Review Articles:** These papers offer a critical perspective on a key body of current research relevant to child and adolescent mental health. The journal requires the pre-registration of review protocols on any publicly accessible platform (e.g. The International Prospective Register of Systematic Reviews, or PROSPERO).

**Short Research Articles:** Short Research Articles should consist of original research of any design that presents succinct findings with topical, clinical or policy relevance. For example, preliminary novel findings from pilot studies, important extensions of a previous study, and topical surveys.

**Letters to the Editor:** These are short articles that offer readers the opportunity to respond to articles published in CAMH. Letters must only discuss issues directly relevant to the content of the original article such as to add context, correction, offer a different interpretation, or extend the findings.

**Innovations in Practice:** These papers report on any new and innovative development that could have a major impact on evidence-based practice, intervention and service models.

**Narrative Matters:** These papers describe important topics and issues relevant to those working in child and adolescent mental health but considered from within the context and framework of the Humanities and Social Sciences.

**Debate Articles:** These papers express opposing points of view or opinions, highlighting current evidence-based issues, or discuss differences in clinical practice.

**Technology Matters:** These papers provide updates on emerging mental health technologies and how they are being used with and by children and young people.

2. Submission of a paper to Child and Adolescent Mental Health will be held to imply that it represents an original submission, not previously published; that it is not being considered for publication elsewhere; and that if accepted for publication it will not be published elsewhere without the consent of the Editors.

3. Manuscripts should be submitted online. For detailed instructions please go to: [http://mc.manuscriptcentral.com/camh.journal](http://mc.manuscriptcentral.com/camh.journal) and check for existing account if you have submitted to or reviewed for the journal before, or have forgotten your details. If you are new to the journal create a new account. Help with submitting online can be obtained from the Editorial Office at ACAMH (email: publications@acamh.org)

4. Authors’ professional and ethical responsibilities

*Disclosure of interest form*
All authors will be asked to download and sign a full Disclosure of Interests form and acknowledge this and sources of funding in the manuscript.
Ethics
Authors are reminded that the journal adheres to the ethics of scientific publication as detailed in the Ethical principles of psychologists and code of conduct (American Psychological Association, 2010). These principles also imply that the piecemeal, or fragmented publication of small amounts of data from the same study is not acceptable. The journal also generally conforms to the Uniform Requirements for Manuscripts of the International Committee of Medical Journal Editors (ICJME) and is also a member and subscribes to the principles of the Committee on Publication Ethics (COPE).

Informed consent and ethics approval
Authors must ensure that all research meets these ethical guidelines and affirm that the research has received permission from a stated Research Ethics Committee (REC) or Institutional Review Board (IRB), including adherence to the legal requirements of the study county. Within the Methods section, authors should indicate that ‘informed consent’ has been appropriately obtained and state the name of the REC, IRB or other body that provided ethical approval. When submitting a manuscript, the manuscript page number where these statements appear should be given.

Preprints
CAMH will consider for review articles previously available as preprints. Authors may also post the submitted version of a manuscript to a preprint server at any time. Authors are requested to update any pre-publication versions with a link to the final published article. Please find the Wiley preprint policy here.

Note to NIH Grantees
Pursuant to NIH mandate, Wiley-Blackwell will post the accepted version of contributions authored by NIH grant-holders to PubMed Central upon acceptance. This accepted version will be made publically available 12 months after publication. For further information, see www.wiley.com/go/nihmandate.

Recommended guidelines and standards
The journal requires authors to conform to CONSORT 2010 (see CONSORT Statement) in relation to the reporting of randomised controlled clinical trials; also recommended is the Extensions of the CONSORT Statement with regard to cluster randomised controlled trials. In particular, authors must include in their paper a flow chart illustrating the progress of subjects through the trial (CONSORT diagram) and the CONSORT checklist. The flow diagram should appear in the main paper, the checklist in the online Appendix. Trial registry name, registration identification number, and the URL for the registry should also be included at the end of the methods section of the Abstract and again in the Methods section of the main text, and in the online manuscript submission. Trials must be registered in one of the ICJME-recognised trial registries:

Australian New Zealand Clinical Trials Registry
Clinical Trials
Netherlands Trial Register
ISRCTN Registry
UMIN Clinical Trials Registry

Manuscripts reporting systematic reviews or meta-analyses will only be considered if they conform to the PRISMA Statement. We ask authors to include within their review article a
flow diagram that illustrates the selection and elimination process for the articles included in their review or meta-analysis, as well as a completed PRISMA Checklist. The journal requires the pre-registration of review protocols on any publicly accessible platform (e.g. The International Prospective Register of Systematic Reviews, or PROSPERO).


CrossCheck
An initiative started by CrossRef to help its members actively engage in efforts to prevent scholarly and professional plagiarism. The journal to which you are submitting your manuscript employs a plagiarism detection system. By submitting your manuscripts to this journal you accept that your manuscript may be screened for plagiarism against previously published works.

5. Manuscripts should be double spaced and conform to the house style of CAMH. The title page of the manuscript should include the title, name(s) and address(es) of author(s), an abbreviated title (running head) of up to 80 characters, a correspondence address for the paper, and any ethical information relevant to the study (name of the authority, data and reference number for approval) or a statement explaining why their study did not require ethical approval.

Summary: Authors should include a structured Abstract not exceeding 250 words under the sub-headings: Background; Method; Results; Conclusions.

Key Practitioner Message: Below the Abstract, please provide 1-2 bullet points answering each of the following questions:

- **What is known?** - What is the relevant background knowledge base to your study? This may also include areas of uncertainty or ignorance.
- **What is new?** - What does your study tell us that we didn't already know or is novel regarding its design?
- **What is significant for clinical practice?** - Based on your findings, what should practitioners do differently or, if your study is of a preliminary nature, why should more research be devoted to this particular study?

Keywords: Please provide 4-6 keywords use MeSH Browser for suggestions

6. Papers submitted should be concise and written in English in a readily understandable style, avoiding sexist and racist language. Articles should adhere to journal guidelines and include a word count of their paper; occasionally, longer article may be accepted after negotiation with the Editors.
7. Authors who do not have English as a first language may choose to have their manuscript professionally edited prior to submission; a list of independent suppliers of editing services can be found at http://authorservices.wiley.com/bauthor/english_language.asp. All services are paid for and arranged by the author, and use of one of these services does not guarantee acceptance or preference for publication.

8. Headings: Original articles should be set out in the conventional format: Methods, Results, Discussion and Conclusion. Descriptions of techniques and methods should only be given in detail when they are unfamiliar. There should be no more than three (clearly marked) levels of subheadings used in the text.

9. All manuscripts should have an Acknowledgement section at the end of the main text, before the References. This should include statements on the following:

**Study funding:** Please provide information on any external or grant funding of the work (or for any of the authors); where there is no external funding, please state this explicitly.

**Contributorships:** Please state any elements of authorship for which particular authors are responsible, where contributorships differ between author group. (All authors must share responsibility for the final version of the work submitted and published; if the study include original data, at least one author must confirm that he or she had full access to all the data in the study and takes responsibility for the integrity of the data in the study and the accuracy of the data analysis). Contributions from others outside the author group should also be acknowledged (e.g. study assistance or statistical advice) and collaborators and study participants may also be thanked.

**Conflicts of interest:** Please disclose any conflicts of interest of potential relevance to the work reported for each of the authors. If no conflicts of interest exist, please include an explicit declaration of the form: "The author(s) have declared that they have no competing or potential conflicts of interest".

10. For referencing, CAMH follows a slightly adapted version of APA Style http://www.apastyle.org/. References in running text should be quoted showing author(s) and date. For up to three authors, all surnames should be given on first citation; for subsequent citations or where there are more than three authors, 'et al.' should be used. A full reference list should be given at the end of the article, in alphabetical order.

References to journal articles should include the authors' surnames and initials, the year of publication, the full title of the paper, the full name of the journal, the volume number, and inclusive page numbers. Titles of journals must not be abbreviated. References to chapters in books should include authors’ surnames and initials, year of publication, full chapter title, editors' initials and surnames, full book title, page numbers, place of publication and publisher.

11. Tables: These should be kept to a minimum and not duplicate what is in the text; they should be clearly set out and numbered and should appear at the end of the main text, with their intended position clearly indicated in the manuscript.
12. Figures: Any figures, charts or diagrams should be originated in a drawing package and saved within the Word file or as an EPS or TIFF file. See [http://authorservices.wiley.com/bauthor/illustration.asp](http://authorservices.wiley.com/bauthor/illustration.asp) for further guidelines on preparing and submitting artwork. Titles or captions should be clear and easy to read. These should appear at the end of the main text.

13. Footnotes should be avoided, but end notes may be used on a limited basis.

**Data Sharing and Supporting Information**

CAMH encourages authors to share the data and other artefacts supporting the results in the paper by archiving them by uploading it upon submission or in an appropriate public repository. Examples of possible supporting material include intervention manuals, statistical analysis syntax, and experimental materials and qualitative transcripts.

1. If uploading with your manuscript please call the file 'supporting information' and reference it in the manuscript.
2. Please note supporting files are uploaded with the final published manuscript as supplied, they are not typeset.
3. On publication your supporting information will be available alongside the final version of the manuscript online.
4. If uploading to a public repository please provide a link to supporting material and reference it in the manuscript. The materials must be original and not previously published. If previously published, please provide the necessary permissions. You may also display your supporting information on your own or institutional website. Such posting is not subject to the journal's embargo date as specified in the copyright agreement. Supporting information is made free to access on publication.

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For information on Sharing and Citing your Research Data see the [Author Services website here](http://authorservices.wiley.com/bauthor/).

**Original Articles**

Original Articles make an original contribution to empirical knowledge, to the theoretical understanding of the subject, or to the development of clinical research and practice. Adult data is not usually accepted for publication unless it bears directly on developmental issues in childhood and adolescence.

Your Original Article should be no more than 5,500 words including tables, figures and references.

**Review Articles**

Research Articles offer our readers a critical perspective on a key body of current research relevant to child and adolescent mental health and maintain high standards of scientific practice by conforming to systematic guidelines as set out in the [PRISMA statement](http://prisma-statement.org/). These
articles should aim to inform readers of any important or controversial issues/findings, as well as the relevant conceptual and theoretical models, and provide them with sufficient information to evaluate the principal arguments involved. All review articles should also make clear the relevancy of the research covered, and any findings, for clinical practice.

Your Review Article should be no more than 8,000 words excluding tables, figures and references and no more than 10,000 including tables, figures and references.

**Short Research Articles**

Short Research Articles should consist of original research of any design that presents succinct findings with topical, clinical or policy relevance. For example, preliminary novel findings from pilot studies, important extensions of a previous study, and topical surveys. Short Research Articles will be peer reviewed and authors might be asked to revise and edit their article to acceptable standards for publication. Short Research Articles should follow standard guidelines, such as STROBE for observational studies, CONSORT extension for pilot trials etc.

Your Short Research Article should be 1500 words, excluding references, tables and graphs/figures. Your article should be structured, including the subheadings Introduction/Methods/Results/Discussion. There is a maximum of 1 table and 1 graph/figure. Please do not include more than 12 references.

**Narrative Matters: The Medical Humanities in CAMH**

These articles are both submissions and directly commissioned papers. They will be peer-reviewed. The articles should be on a humanities topic relevant to those working in child and adolescent mental health. The topics can include but are not restricted to: aspects of child mental health service history; representations of abnormal mental states or mental illness in children and teenagers in film, literature or drama; depictions of child mental health clinicians within popular culture; ethical dilemmas in the speciality. Interest and originality are valued. If in doubt, please contact the section editor: Gordonbates@virginmedia.com

The essays should be between 1500 and 2000 words and written for an audience of child mental health professionals. For publishing reasons, there is an upper limit of 8 references for the article. Additional references may be given in the text if necessary.

**Letters to the Editor**

Letters to the Editor are short articles that offer readers the opportunity to respond to articles published in CAMH. Letters must only discuss issues directly relevant to the content of the original article such as to add context, correction, offer a different interpretation, or extend the findings. Letters will be evaluated for relevance to the index paper, scientific merit, and importance.

Letters should be submitted not later than 2 weeks after publication of the print issue of the Journal containing the paper of interest. Please note - all papers are published on Early View as soon as they are accepted. The letters should avoid personal attacks and unscholarly communication.
Letters will not be peer reviewed. However, the section Editor will review the letters and might consult another Editor before acceptance or rejection.

Due to the short length of this article type, your Letter should be between 500 and 700 words with a maximum of one figure or table. If in doubt, please contact the section editor c.ani@imperial.ac.uk

**Innovations in Practice**

Innovations in Practice promote knowledge of new and interesting developments that have an impact on evidence-based practice, intervention and service models. These might have arisen through the application of careful, systematic planning, a response to a particular need, through the continuing evolution of an existing practice or service, or because of changes in circumstances and/or technologies. Submissions should set out the aims and details of the innovation including any relevant mental health, service, social and cultural contextual factors, and give a close, critical analysis of the innovation and its potential significance for the practice of child and adolescent mental health.

Due to the short length of this article type, your Innovations in Practice article should be no more than 2,200 words including tables, figures and references and contain no more than 8 references.

**Debate Articles**

Our debate articles express opposing points of view or opinions, highlighting current evidence-based issues, or discuss differences in clinical practice. Although discussion of evidence is welcome, these articles generally do not include primary data. The evidence on which your arguments are based and how that was sourced should be explicit and referenced, and the quality of your evidence made clear.

Due to the short length of this article type, your Debate article should be no more than 1,000 words and contain no more than 8 references. If in doubt, please contact the section editor Rachel.Elvins@mft.nhs.uk

**Technology Matters**

Technology Matters provides updates on emerging mental health technologies and how they are being used with and by children and young people. We aim to cover established technologies such as computer-assisted psychological interventions as well as more novel technologies (e.g. mobile apps, therapeutic games, virtual reality). We will present the evidence base for their use, showcase how they can complement other interventions and are being used in practice and address wider cross-cutting issues (such as technology accreditation, regulation, cost etc.) relevant to practitioners and service funders.

Your paper should be between 1000 and 1500 words. Please do not include more than 7 references. If in doubt, please contact the section editors Kapil.Sayal@nottingham.ac.uk or Jennifer.Martin@nottingham.ac.uk.

**Manuscript Processing**

**Peer Review Process:** All material submitted to CAMH is only accepted for publication after being subjected to external scholarly peer review, following initial evaluation by one of the Editors. Both original and review-type articles will usually be single-blind reviewed by a minimum of two external referees and only accepted by the decision Editor after
satisfactory revision. Any appeal of an editorial decision will first be considered by the initial decision Editor, in consultation with other Editors. Editorial practices and decision making will conform to COPE http://publicationethics.org/resources/guidelines and ICMJE http://icmje.org/ best practice.

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In accordance with Wiley's Best Practice Guidelines on Research Integrity and Publishing Ethics and the Committee on Publication Ethics' guidance, CAMH will allow authors to correct authorship on a submitted, accepted, or published article if a valid reason exists to do so. All authors – including those to be added or removed – must agree to any proposed change. To request a change to the author list, please complete the Request for Changes to a Journal Article Author List Form and contact either the journal's editorial or production office, depending on the status of the article. Authorship changes will not be considered without a fully completed Author Change form. Correcting the authorship is different from changing an author's name; the relevant policy for that can be found in Wiley's Best Practice Guidelines under “Author name changes after publication.”

Wiley's Author Name Change Policy
In cases where authors wish to change their name following publication, Wiley will update and republish the paper and redeliver the updated metadata to indexing services. Our editorial and production teams will use discretion in recognizing that name changes may be of a sensitive and private nature for various reasons including (but not limited to) alignment
with gender identity, or as a result of marriage, divorce, or religious conversion. Accordingly, to protect the author's privacy, we will not publish a correction notice to the paper, and we will not notify co-authors of the change. Authors should contact the journal's Editorial Office with their name change request.

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Appendix F: Pre-registration of Hypotheses, Methods, and Analyses for Paper 2

Preregistration

July 1, 2021

1 Study information

1.1 Title
Disentangling the relationships between motor functioning and cognitive control in young children with symptoms of ADHD.

1.2 Authors
Cameron Ferguson, Christopher Hobson, Stephanie Van Goozen, Kate Anning, and Craig Hedge.

1.3 Description
Children with ADHD display difficulties with both motor and cognitive control. This is unsurprising given the emergence of cognitive control from motor control in childhood (Koziol et al., 2013; Koziol et al., 2012) and the central role of frontostrriatal and cerebellar circuits in the control of thought as well as movement (Diamond, 2013; Frank & Badre, 2015). However, while relationships between elements of motor and cognitive control are apparent, the exact nature of these remains unclear. Motor control can refer to a wide array of abilities, including generating consistent motor output and controlling movement in relation to visual feedback in situations with varying planning demands. Similarly, cognitive control is an umbrella term. It can be separated into at least three components in young children: inhibition (i.e., withholding automatic responses), switching (i.e., change focus from task to task), and working memory monitoring and updating (i.e., keeping track of and refreshing the information held in the mind’s eye) (Henry & Bettenay, 2010; Miyake et al., 2000). The available evidence suggests that motor control is most frequently associated with cognitive inhibition (Mostofsky et al., 2003; Rigoli et al., 2012) in children with ADHD, although there is also some evidence of links between motor control and working memory (Rigoli et al., 2012). However, given the (partially) overlapping neural circuitry, it is reasonable to suggest that other aspects of cognitive control (e.g., switching/flexibility) are also linked to motor control. Overall, it is unclear which aspects of cognitive control are associated with motor abilities in children presenting with symptoms of ADHD.

One reason for the lack of clarity regarding the relationship between motor and cognitive control in understanding ADHD symptomatology is that common measures of inhibition, switching and working memory updating are not-process specific, despite purportedly measuring a particular element of cognitive control. For example, performance on the flanker task, which at face value measures a unitary ability to inhibit a prepotent response, is multidetermined (Sergeant, 2005). Rather than being a simple reflection of inhibitory control, flanker performance appears to depend on how quickly an individual can gather information to inform their decision, how sure they tend to be before they commit to a response, and how long it takes them to encode stimuli and prepare for motor actions (Ratcliff & McKoon, 2008). By
separating composite variables into subcomponents, cognitive modelling may facilitate the disentangling of the relationships between motor and cognitive control in children with varying levels of hyperactive-impulsive ADHD symptomatology.

This two-part study aims to clarify the relationship between motor and cognitive control in a relatively large sample of young children with varying levels of hyperactive-impulsive ADHD symptomatology. Part 1 of the study will investigate which aspects of motor control (motor generation and/or visuomotor control in the context of tasks with low and high planning demands) predict which aspects of cognitive control (inhibition, switching/flexibility, and/or working memory) and whether these relationships are moderated by hyperactive-impulsive symptom severity. Part 2 of the study will use cognitive modelling to indicate how motor control might influence performance on a cognitive inhibition task in terms of the constituent processes required for task success (or failure).

In part 1 of the study, it is anticipated that cognitive inhibition will be predicted by motor generation and visuomotor control with low planning demands. Specifically, it is predicted that children with greater motor output and better visuomotor control in a low planning demand context will perform better on the flanker task. This is because the measure of cognitive inhibition employed, a flanker task, is a speeded two alternative forced-choice paradigm requiring fast responses with little time for careful, deliberate response planning and thus greater motor output may be helpful for ensuring quick responses. Additionally, given the interference control demands of the flanker task, it is suggested that greater ability to control visually-guided movement in the absence of high planning demands will also be predictive of better flanker performance as children will have to display focused attention and consistently perform accurately despite the speed of the task.

It is also expected that, in moderation analysis, children with greater levels of hyperactive-impulsive ADHD symptomatology will display a relationship between motor generation and flanker performance in the opposite direction. That is, for children with high levels of hyperactivity-impulsivity, greater motor output will be associated with poorer cognitive inhibition. This prediction is made based on clinical observations and theories suggesting that there is an optimal window of arousal and activation regarding cognitive task performance for children with ADHD (Sergeant, 2005).

Regarding foundational working memory abilities (i.e., capacity and maintenance), it is predicted that motor generation will be positively associated with both verbal and visuospatial working memory domains. This is in light of the persistence and consistency involved in performing the tapping task (motor generation) successfully and the assumption that the successful maintenance of information in working memory following acquisition is a process requiring persistence and effort akin to prolonged motor generation over time. It is also anticipated that the positive association between motor output and working memory will be reversed in children with higher levels of hyperactivity-impulsivity. This is because higher motor activity is usually understood as pathological and related to cognitive impairments in ADHD.

Regarding working memory processing (i.e., manipulation), it is anticipated that visuomotor control in both low- and high-planning contexts will be predictive. This is because individual differences in manipulatory working memory processes in childhood might be (partly) explained by individual differences in continuously adjusting movements based on visual feedback (i.e., visuomotor control) in situations regardless of whether intensive planning is possible. Additionally, it is expected that children with high hyperactive-impulsive symptoms and poor visuomotor control will perform even more poorly on a measure of working memory manipulation.

With regards to switching/flexibility, given the multifaceted nature of this construct and the many functions tapped by the tasks used to measure it (which include set maintenance and
inhibitory processes regarding withholding previously positively reinforced responses as well as attention switching/flexibility per se (Cragg & Chevalier, 2012), it can be speculated that motor generation and visuomotor control in both low and high planning contexts will be positively associated with performance on the task. It is also anticipated that children with high levels of hyperactive-impulsivity who display greater motor output will perform less well on switching than their low-hyperactive-impulsive peers.

In part 2 of the study, it is anticipated that motor generation is predictive of the non-decision time element of performance on a flanker task, with children who demonstrate greater motor output displaying shorter non-decision time estimates. This prediction is made in light of the assumption that nondecision time in the drift diffusion partly reflects motor preparation (as well as stimuli encoding). While moderation by hyperactive-impulsive ADHD symptoms will be tested, a directional prediction is not made. Slower (and more variable reaction times) have been linked with ADHD. As minimum reaction times shape the non-decision time parameter estimate in the DDM, this implies larger (i.e., slower) nondecision time parameter estimates for children with higher ADHD symptomatology. However, it is also plausible that ‘hyperactivity’ is present in motor output for children with high hyperactive-impulsive symptoms. This would lead to reduced nondecision time parameter estimates. Indeed, existing research is unequivocal. One study suggests that a group of children aged 6-17 with ADHD displayed faster non-decision time than a healthy control group on a simple perceptual decision-making task and an executive control task (Metin et al., 2013). However, another study did not find any differences in non-decision time across comparable ADHD and healthy control groups (Karalunas et al., 2012).

Finally, it is predicted that visuomotor control in low-planning contexts is related to boundary separation estimates. Children who are better able to control fine motor movements in response to visual stimuli might reasonably display more conservative response strategies (i.e., larger boundary separation estimates) on speeded two-alternative forced-choice reaction time tasks. Alternatively, children might display negative associations between low-planning visuomotor control and boundary separation. This is because individuals with greater ability can benefit from a liberal response strategy (i.e., smaller boundary separation estimates), as they do not need to continue to acquire unnecessary evidence to make accurate decisions (Schmiedek et al., 2007). Accordingly, a non-directional prediction is offered.

Again, while moderation effects regarding hyperactive-impulsive symptoms will be investigated, a directional prediction is not offered. This is because although slow and variable response times on speeded reaction time tasks have been observed in children with ADHD, which would suggest larger boundary separation in line with a speed-accuracy trade-off favouring accuracy, accuracy is also usually poor, which would suggest suboptimal boundary separation (and/or drift rate). Moreover, there is a paucity of research specifically using the flanker task as opposed to other measures of inhibitory control in children with ADHD, which prevents strong predictions from being offered.

Slow drift rate (i.e., the speed at which information is acquired) is reliably associated with ADHD in children (Feldman & Huang-Pollock, 2021; Haller et al., 2021; Huang-Pollock et al., 2017, 2020; Karalunas et al., 2012). It is anticipated that drift rate will be predicted by motor generation and visuomotor control in a low-planning context, and that this relationship will be moderated by hyperactive-impulsive ADHD symptoms as the cognitive-energetic model of ADHD (Sergeant, 2005) predicts a quadratic association between ‘activation’ (i.e., tonic changes of physiological activity) and ‘arousal’ (i.e., phasic responding time-locked to stimulus processing) and performance on cognitive tasks.
2 Hypotheses

2.1 Part 1: which elements of cognitive control are predicted by motor generation and/or visuomotor control?

1. Children who display greater motor output and visuomotor control in a low planning demand task will display better cognitive inhibition (one-tailed).

2. Children with higher levels of hyperactive-impulsive ADHD symptomatology will display a negative association between motor generation and cognitive inhibition (one-tailed).

3. Children who display greater motor output will display better verbal and visuospatial working memory (one-tailed).

4. Children with higher levels of hyperactive-impulsive ADHD symptomatology will display a negative association between motor generation and working memory (one-tailed).

5. Children who display greater visuomotor control will be better at visuospatial working memory manipulation (one-tailed).

6. Children who display poorer visuomotor control who display high levels of hyperactive-impulsive symptomatology will perform even more poorly on an index of visuospatial working memory manipulation (one-tailed).

7. Children who display greater motor output and better visuomotor control in low and high planning paradigms will display greater switching ability (one-tailed).

8. Children with higher levels of ADHD symptomatology will display a negative association between motor generation and switching ability (one-tailed).

2.2 Part 2: which processes contributing to flanker performance are predicted by motor generation and/or visuomotor control?

1. Children with greater motor output will display reduced non-decisional processing time on a drift-diffusion model of flanker performance (one-tailed).

2. The relationship between motor generation and non-decisional processes is moderated by hyperactive-impulsive ADHD symptom severity (two-tailed).

3. Visuomotor control in a low planning demand paradigm is related to boundary separation estimates in a drift-diffusion model of flanker performance (two-tailed).

4. The relationship between low planning demand visuomotor control and boundary separation is moderated by hyperactive-impulsive ADHD symptom severity (two-tailed).

5. Motor output and visuomotor control in a low-planning task will be predictive of drift rate (two-tailed).

6. The relationship between motor output and low-planning visuomotor control and drift rate will be moderated by hyperactive-impulsive-ADHD symptom severity (two-tailed).
3 Design plan
3.1 Study type
Observational study.

3.2 Blinding
The lead researcher (C. Ferguson) was not involved in assessment of the participants. Otherwise, no blinding is involved in the study.

3.3 Study design
Cross-sectional, between-subjects design.

3.4 Randomisation
N/a.

4 Sampling plan
4.1 Registration prior to analysis of the data
The data exist and have been accessed to check the labelling and missing values for the variables of interest to the study. No analysis (including the calculation of summary statistics) has been conducted related to the research plan.

There are several publications associated with the database for the Neurodevelopmental Assessment Unit. The lead researcher for the current study (C. Ferguson) and supervisor (C. Hedge) was not involved with any of these projects in any way, although the main supervisors (C. Hobson and S. Van Goozen) and the co-author (K. Anning) have been. Data for the motor control variables (i.e., the Amsterdam Neuropsychological Tasks Tapping, Tracking and Pursuit tasks) have not been analysed before.

4.2 Data collection procedures
Data were collected from children aged 4- to 11-years-old with diverse presenting problems who were referred to the Neurodevelopmental Assessment Unit at Cardiff University. Recruitment was primarily from schools across South East Wales, who referred children for assessment. Children received a small gift in exchange for participating. The referrer received a report describing the child's cognitive strengths/weaknesses and recommending compensatory strategies.

4.3 Inclusion/exclusion criteria
Children will be excluded from analyses if their estimated general cognitive functioning falls below a scaled score of 70 (where mean = 100, standard deviation = 15) on the Lucid Ability Test. This is to help ensure that the study is investigating individual differences in motor and cognitive control ability rather than the effects of very low general cognitive ability and possible intellectual disability.

Children will be excluded from an analysis if they are missing data for the variables of interest to that analysis. No data imputation strategy will be used.
4.4 Sample size

At present (July 1, 2021), the database contains data for around 300 children, but many of the children struggle to complete some tests. Hence the likely available sample size will be around 200. Application of the exclusion criteria is likely to reduce this further.

4.5 Sample size rationale

A sample size of at least 153 participants is needed to confer at least 80% power to detect a relatively small relationship of $f^2 = 0.065$ between motor generation/control variables and cognitive control variables on the basis of the inclusion of seven predictors in a linear multiple regression model with R2 increase. The expected effect size is taken from Rigoli et al. (2012) who observed that motor control significantly predicted a small portion of the variance in performance on a test of inhibition ($p = .017$, sr2 = .061, equivalent to $f^2 = .065$) in a sample of adolescents.

5 Variables

5.1 Measured variables

5.1.1 Inclusion/exclusion variables Lucid Ability Test

The Lucid Ability Test provides an estimate of 'intelligence' or general cognitive ability for children based on performance across a picture vocabulary task and a mental rotation task. The score is age-corrected by reference to normative data. As previously mentioned, the Lucid Ability Test will be used to excluded participants whose estimated general cognitive function in the very low range range (≤ 69).

5.1.2 Predictor variables Amsterdam Neuropsychological Tasks Tapping

The Amsterdam Neuropsychological Tasks (ANT) Tapping task is a measure of self-generated motor output without internal or external cues. The task generates a Z score for the number of taps generated, which is referenced to an age-stratified normative sample. Norm-referenced data ensures that each child's motor output is placed in the context of what is typical for their age. Data for the dominant hand will be used.

ANT Tracking

ANT Tracking is a test of visuomotor control with low planning demands which provides Z scores for accuracy (i.e., the mean distance from the midline averaged across equal-sized segments of the circle) and variability (i.e., the standard deviation of the mean distance from the midline averaged across equal-sized segments of the circle) of movement. Data for the dominant hand will be used. In the event of high correlation (i.e., r ≥ 0.80) between the mean distance and standard deviation of the mean distance, the mean distance score will be entered into regression analyses (described below) to avoid the problem of multicolinearity.

ANT Pursuit

ANT Pursuit is a test of visuomotor control with high planning demands which provides Z scores for accuracy (i.e., the mean distance from the trajectory of a target moving in an unpredictable manner) and variability (i.e., the standard deviation of the mean distance from the trajectory of
a target moving in an unpredictable manner) of movement. Data for the dominant hand will be used. In the event of high correlation (i.e., $r \geq 0.80$) between the mean distance and standard deviation of the mean distance (which is plausible as the standard deviation is influenced by the mean), the mean distance score will be entered into regression analyses (described below) to avoid the problem of multicolinearity.

5.1.3 Covariates Development and Wellbeing Assessment ADHD

Hyperactive Impulsive Symptom Score

A standardised assessment of child mental health problems. The hyperactive impulsive score of the ADHD scale will be used as a covariate in the multiple regression analyses described below. The inattentive score of the ADHD scale will be presented in descriptive statistics to characterise the sample but will not be used in any inferential analysis.

5.1.4 Dependent variables

National Institute of Health Toolbox Flanker

The National Institute of Health (NIH) Toolbox Flanker is a test of cognitive inhibition. Unlike the traditional reaction time cost index of flanker performance (i.e., incongruent reaction time minus congruent reaction time), the NIH Flanker task provides a single combined score for accuracy and, for participants who achieve more than 80% accuracy, reaction times. Children aged 3-7 years are initially presented with 20 trials of fish stimuli (12 congruent, 8 incongruent). If a child aged 3-7 scores $\geq 90\%$ on the fish stimuli, 20 additional trials with arrows are presented (12 congruent, 8 incongruent). In the NDAU sample, most participants complete 40 trials (20 fish plus 20 arrow). This score is age-corrected by reference to normative data stratified by year of age. Children aged 8+ are presented with 20 trials of arrow stimuli (12 congruent, 8 incongruent). Individual trial data for decision and reaction time are also available as output from the computer program and these will be used for cognitive modelling (see below).

NIH Toolbox Dimensional Change Card Sort

The NIH Toolbox Dimensional Change Card Sort (DCCS) task is a test of shifting. The DCCS task provides a single combined score for accuracy and, for participants who achieve more than 80% accuracy, reaction times. This score is age-corrected by reference to normative data stratified by year of age.

Automated Working Memory Assessment Backwards Digit Recall

The Automated Working Memory Assessment (AWMA) Backwards Digit Recall is a test of verbal working memory. The score is age-corrected by reference to normative data stratified by year of age.

AWMA Mister X

A test of visuospatial working memory. Two metrics are provided, an accuracy score (which reflects foundational visuospatial working memory abilities such as capacity and maintenance) and a processing score (which reflects the manipulation aspect of visuospatial working memory). Each score is expressed as a standard score ($\text{mean} = 100, \text{SD} = 15$) which is age-corrected by reference to normative data stratified by year of age.
Drift-diffusion model parameters

The non-decision time and boundary separation parameter estimates from the drift diffusion model will be used as dependent variables. For more information, see ‘Cognitive modelling’ section below.

6 Cognitive modelling

6.1 Cognitive model

In Part 2 of the study, EZ DM will be used to estimate a basic drift-diffusion model (DDM) of performance on the NIH Toolbox Flanker task. The DDM is a cognitive model which assumes that, while making a binary decision (e.g., whether to click left or right on a flanker task), information is continuously sampled, in a noisy diffusion-like process, from the displayed stimulus until enough evidence has accumulated to make a response. A response is initiated once one of two thresholds has been reached. The nature of the response (i.e., whether it is correct or incorrect) depends on which threshold was hit during the decision process.

Unlike the parameters of statistical models, the parameters of the DDM a cognitive model - are psychologically meaningful (Busemeyer & Diederich, 2010). Drift rate sets the average slope of the diffusion process and reflects the speed at which information is acquired. Boundary separation refers do the distance between the two decision thresholds. Larger values lead to longer decision processes, on average, whereas smaller values lead to shorter decision processes, on average. In cognitive terms, a larger boundary separation value implies a more conservative decision making style as more evidence is needed for a decision to be made while a smaller boundary separation value suggests that the participant requires less information to make a decision. It is associated with speed-accuracy trade-off. Non-decision time refers to the time before and after the decision process (which is characterised by drift rate and boundary separation) and reflects the time needed for stimuli encoding and motor preparation (Ratcliff & McKoon, 2008).

The EZ DM method estimates values for drift rate, boundary separation, and non-decision time parameters based on mean reaction time, the variance of reaction time, and accuracy data (i.e., accuracy coding will be used for the DDM.) Congruent and incongruent trials will be modelled simultaneously, given the low trial numbers (40) and the focus on non-decision time.

6.2 Data inclusion/exclusion

Only data for participants who completed the full 40 trial flanker task (24 congruent and 16 incongruent trials) will be used for cognitive modelling as this number of trials is already very low. Participants who completed a 20-trial version of the flanker task (fish-only or arrow-only stimuli) will not be included in cognitive modelling or related statistical analyses. As in Haller et al. (2021), trials featuring non-physiologic anticipation responses (RT ≤ 150ms) will be excluded. The NIH Flanker employs a 10 second cut-off for responses to trials; however, as the EZ DM method is sensitive to slow contaminants (Ratcliff, 2008), slow responses of ≥ 3 seconds will be excluded. An accuracy criterion for inclusion will not be used as the NIH Flanker requires 90% accuracy to progress from fish stimuli trials to arrow stimuli trials and therefore be eligible for inclusion (i.e., have completed 20 fish plus 20 arrow trials) for cognitive modelling.
6.3 Robustness checks

A parameter recovery approach will be used to investigate if the model estimation can consistently identify different levels of a parameter in the data, as in Hedge et al. (2020). This check can be described in terms of a four-step process. First, the model will be estimated and parameter estimates extracted for each participant. Second, data will be simulated under these parameters for each participant using the \textit{construct-samples} program associated with \textit{fast-dm} (Voss & Voss, 2007). Third, the model will be fit to the simulated data using the same procedure that was used in step one. Finally, the parameter estimates from step one and step four will be correlated with Pearson’s correlations. Higher values are indicative of better parameter recovery.

7 Statistical models

Note that all planned analyses are denoted in the way in which they will be entered as input to the statistical models in R. For example, the code “Tapping Z * DAWBA HI Symptoms”, will cause the testing of Tapping Z, DAWBA HI Symptoms and Tapping Z * DAWBA HI symptoms (moderation) predictors in a multiple linear regression.

As previously mentioned (see ‘Predictor variables’ section) SD scores for Tracking and Pursuit tasks will be dropped from all analyses if they are highly correlated with other variables to guard against multicollinearity.

The assumptions of normally distributed residuals, no multicollinearity and homoscedasticity will be tested before the results of the multiple linear regression analyses are viewed. In the event of non-normally distributed residuals, transformation will not be used to preserve the interpretability of the linear models, although results will be interpreted with greater caution. The Mahalanobis Distance test with an alpha level of .001 will be used to test for multivariate outliers, which will be removed if found.

The directional and non-directional hypotheses to be tested in each analysis are denoted below; p values will be halved for tests of directional hypotheses.

7.1 Part 1: which elements of cognitive control are predicted by motor generation and/or visuomotor control?

Multiple linear regression analysis (shown in Table 1) will be used to test the directional hypotheses \textit{children who display greater motor output and visuomotor control in a low planning demand task will display better cognitive inhibition and children with higher levels of hyperactive-impulsive ADHD symptomatology will display a negative association between motor generation and cognitive inhibition}. As both hypotheses are directional and multiple linear regression in R is two-tailed by default, the resulting p values for the predictor (hypothesis 1) and the moderation term (hypothesis 2) in the model will be divided by two.

Post-hoc simple slope analysis will be used to probe the hypothesised moderation effect, if present. As previously mentioned, the Tracking SD score will not be included in the model if it is highly correlated with the Tracking Mean score to avoid multicollinearity. Before the results are presented, the assumptions of the model will be checked.

Multiple linear regression will also be used to test the hypotheses \textit{children who display greater motor output will display better verbal and visuospatial working memory and children with higher levels of hyperactive-impulsive ADHD symptomatology will display a negative association between motor generation and working memory}. The models that will be used to test these hypotheses are shown in Table 2 and Table 3. Again, as these hypotheses are directional, the
relevant P values will be halved. Post-hoc simple slopes analysis will be used to probe the hypothesised moderation effect.

Table 1: Multiple linear regression analysis to test part 1 hypotheses 1 and 2.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Dependent variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tapping Z * DAWBA HI Symptoms</td>
<td>Flanker Age-Corrected Score</td>
</tr>
<tr>
<td>Tracking Mean Z * DAWBA HI Symptoms</td>
<td></td>
</tr>
<tr>
<td>Tracking SD Z * DAWBA HI Symptoms</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Multiple linear regression analysis to test part 1 hypotheses 3 and 4.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Dependent variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tapping Z * DAWBA HI Symptoms</td>
<td>Backwards Digit Recall (age-corrected)</td>
</tr>
</tbody>
</table>

Table 3: Multiple linear regression analysis to test part 1 hypotheses 3 and 4.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Dependent variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tapping Z * DAWBA HI Symptoms</td>
<td>Mister X (age-corrected)</td>
</tr>
</tbody>
</table>

Multiple linear regression (shown in Table 4) will also be used to test the directional hypotheses that *children who display greater visuomotor control will be better at visuospatial working memory manipulation* and *children who display poorer visuomotor control who display high levels of hyperactive impulsive symptomatology will perform even more poorly on an index of visuospatial working memory manipulation*. Again, as these hypotheses are directional, the relevant p values will be halved. Post-hoc simple slopes analysis will be used to probe the hypothesised moderation effect, if present.

Finally, multiple linear regression will also be used to test the hypotheses that *children who display greater motor output and better visuomotor control in low and high planning paradigms will display greater switching ability* and *children with higher levels of ADHD symptomatology will display a negative association between motor generation and switching ability*. The model is shown in Table 5. As these hypotheses are directional, the relevant P values will be halved. Post-hoc simple slopes analysis will be used to probe the hypothesised moderation effect, if present.

Table 4: Multiple linear regression analysis to test part 1 hypotheses 5 and 6.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Dependent variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tracking Z * DAWBA HI Symptoms</td>
<td>Mister X processing (age-corrected)</td>
</tr>
<tr>
<td>Tracking Z SD * DAWBA HI Symptoms</td>
<td></td>
</tr>
<tr>
<td>Pursuit Z * DAWBA HI Symptoms</td>
<td></td>
</tr>
<tr>
<td>Pursuit Z SD * DAWBA HI Symptoms</td>
<td></td>
</tr>
</tbody>
</table>

Table 5: Multiple linear regression analysis to test part 1 hypotheses 7 and 8.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Dependent variable</th>
</tr>
</thead>
</table>
7.2 Part 2: which processes contributing to flanker performance are predicted by motor generation and/or visuomotor control?

As shown in Table 6, multiple linear regression will be used to test the directional hypothesis that *children with greater motor output will display reduced non-decisional processing time on a drift-diffusion model of flanker performance* and the non-directional hypothesis that *the relationship between motor generation and non-decisional processes is moderated by hyperactive impulsive ADHD symptom severity*. Post-hoc simple slope analysis will also be used to probe the hypothesised moderation effect, if present.

Table 6: Multiple linear regression analysis to test part 2 hypotheses 1 and 2.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Dependent variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tapping Z * DAWBA HI Symptoms</td>
<td>Non-decision time (DDM)</td>
</tr>
</tbody>
</table>

Multiple linear regression, displayed in Table 7, will also be used to test the directional hypothesis that *children who display greater visuomotor control in a low planning demand paradigm will display greater boundary separation on a drift-diffusion model of flanker performance* and the non-directional hypothesis that *the relationship between low planning demand visuomotor control and boundary separation is moderated by hyperactive impulsive ADHD symptom severity*. Post-hoc simple slope analysis will also be used to probe the hypothesised moderation effect, if present.

Table 7: Multiple linear regression analysis to test part 2 hypotheses 3 and 4.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Dependent variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tracking Z * DAWBA HI Symptoms</td>
<td>Boundary separation (DDM)</td>
</tr>
<tr>
<td>Tracking SD * DAWBA HI Symptoms</td>
<td></td>
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</tbody>
</table>

Finally, multiple linear regression will be used to test the two-tailed hypotheses that *motor output and visuomotor control in a low-planning task will be predictive of drift rate* and *the relationship between motor output and low-planning visuomotor control and drift rate will be moderated by hyperactive-impulsive-ADHD symptom severity*.

Table 8: Multiple linear regression analysis to test part 2 hypotheses 5 and 6.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Dependent variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tapping Z * DAWBA HI Symptoms</td>
<td>Drift rate (DDM)</td>
</tr>
<tr>
<td>Tracking Z * DAWBA HI Symptoms</td>
<td></td>
</tr>
<tr>
<td>Tracking SD * DAWBA HI Symptoms</td>
<td></td>
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</tbody>
</table>
7.3 Inference Criteria

An alpha level of .05 will be used as a threshold for considering results statistically significant. As multiple tests enable the rejection of the null hypothesis relating to hypotheses 3 and 4 and hypotheses 5 and 6 in part 1 of the study and regarding hypotheses 5 and 6 in part 2 of the study, the regression coefficients in each relevant model will be subject to Holm-Bonferroni correction for multiple comparisons to control for the elevated type I error rate.

Only confirmatory analyses will be subject to correction for multiple as the type I error rate cannot be estimated for exploratory analyses (Nosek & Lakens, 2014). Inferences will not be drawn from exploratory analyses although hypotheses may be generated for future confirmatory research.

7.4 Missing data

Subjects with missing data will be removed from each relevant analysis. Missing data imputation strategies will not be used.

7.5 Exploratory analyses

All exploratory analyses will be reported as such and inferences regarding the population of interest will not be made.

7.5.1 Part 1: which elements of cognitive control are predicted by motor generation and/or visuomotor control?

Multiple linear regression analyses, with post-hoc simple slope analysis if applicable, may be used to explore whether any dependent variable is predicted by any combination of motor generation and/or visuomotor control with low and/or high planning demands variables, and whether any relationship is moderated by hyperactive-impulsive and/or inattentive ADHD symptom severity. Two-tailed tests of hypothesised effects in the opposite direction and the influence of age and gender may also be explored.

7.5.2 Part 2: which processes contributing to anker performance are predicted by motor generation and/or visuomotor control?

Multiple linear regression analyses may be used to explore whether motor generation and/or visuomotor control with low and/or high planning demands predict the drift rate, nondecision time or boundary separation DDM parameters for congruent and incongruent trials. Post-hoc simple slopes analyses may be used to probe any moderation effects. Two-tailed tests of hypothesised effects in the opposite direction and the influence of age and gender may also be explored.

References


Appendix G: Statement of Ethical Approval for Paper 2

From: psychethics <psychethics@cardiff.ac.uk>
Subject: Ethics Feedback - EC.16.10.11.4592GRA5
Date: 5 July 2018 at 10:34:22 BST
To: Stephanie Van Goozen <VangoozenS@cardiff.ac.uk>

Dear Steph,

The Ethics Committee has considered the amendment to your Staff project proposal: A Feasibility Study of a Neurodevelopmental Disorders Assessment Unit (EC.16.10.11.4592GRA5).

The amendment has been approved on the condition that a comment is added to the information, stating that if a child shows distress the monitor can be removed immediately.

Please note that if any changes are made to the above project then you must notify the Ethics Committee.

Best wishes,
Mark Jones

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Appendix II: Parental Consent Form for Paper 2

STUDY CONSENT FORM
(for parents of children aged 4-7 years)
This is to be completed by parents/care-givers on behalf of their child and themselves.

1. I confirm that I have read and understood the information sheet for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

2. I understand that my participation and that of my child is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights being affected.

3. I am happy for the research team to make contact with me if there are any future research studies that might be of interest to me.

4. I agree for my child to perform the developmental assessments as part of the study named above, including measuring my child’s heart-rate.

5. I agree to complete the parental interview and questionnaires as part of the study named above.

6. I understand that relevant sections of my child’s data collected during the study (including my ratings about my child on the Strengths and Difficulties Questionnaire) may be looked at by individuals from the NDAU study team, from regulatory authorities or by my child’s referring agent, where it is relevant to their taking part in this research. I give permission for these individuals to have access to my child’s data.

7. I understand that an assessment report of my child’s strengths and difficulties will be sent to the referring agent to guide their intervention with my child within the school environment. I understand that I do not receive a copy of this report.

8. I understand that a video recording will be made of my child’s assessments for research, safety and training purposes. I understand that brief clips from the video may be used to illustrate important aspects of child development, and to train new researchers, and so such clips may be shown to students or at professional meetings. I give consent for such clips to be taken from this video record, with the understanding that my name or my child’s name will never be associated with the video clip. I understand that the video will remain in the possession of Prof. Van Goozen and the NDAU research team, and will never be given to other unauthorised individuals.
9. I agree that assessment can be linked to routinely collected, anonymised datasets (such as those held in the Secure Anonymised Information Linkage [SAIL] databank), in order to answer future questions related to mental health. I understand that the data within any such dataset will be fully anonymised and my child would not be identifiable in any way.

____________________  __________
Name of parent     Date     Signature

____________________  __________
Name of person taking consent  Date     Signature

The information provided will be held in compliance with GDPR regulations. Cardiff University is the data controller and Matt Cooper is the data protection officer (inforequest@cardiff.ac.uk). The lawful basis for processing this information is public interest. This information is being collected by Professor Stephanie van Goozen. The information on the consent form will be held securely and separately from the research information. Only the researcher will have access to this form and it will be destroyed after 7 years. The research information you provide will be used for the purposes of research only and will be stored securely. Only members of the NDAU research team will have access to this information. After 7 years the data will be anonymised (any identifying elements removed) and this anonymous information may be kept indefinitely or published.
Appendix I: Additional Descriptive Statistics for Paper 2

The pre- and post-transformation distributions of all variables are presented in Figures 6 and 7. Outliers, which were removed prior to data transformation, are visible in Tracking (z scores of -22.4 and -22.5) and Pursuit (z score of -63.0) distributions in Figure 6.

Figure 6. Pre-transformation Distributions of Variables.

Figure 7. Post-transformation Distribution of Variables.

Appendix J: Robustness Checks for Cognitive Modelling in Paper 2

Three robustness checks were performed to test relative fit, absolute fit, and prediction of Flanker performance. The parameter recovery routine was performed, as in Hedge et al. (2020), to examine the relative fit of the EZ-DM parameter estimates. This routine followed a four-stage process. First, the EZ-DM parameter estimates for each participant were extracted. Second, the construct-samples programme (Voss & Voss, 2007) was used to simulate data for each child’s empirical EZ-DM parameter estimates. Third, EZ-DM was used to estimate parameter values for each child’s artificial data generated in step 2. Finally, the EZ-DM parameter estimates for the original, empirical participant data and the synthetic participant data arising from step three were correlated. Results are shown in Figure 8.

Figure 8. Cognitive Modelling Robustness Check Parameter Recovery Results.

Additionally, the means of the empirical and simulated reaction time data were compared with t tests to probe the absolute fit of the EZ-DM modelling. These results, which are displayed in Table 6, imply adequate absolute fit as the empirical and synthetic reaction times were not significantly different.
Table 6. Comparison of Empirical and Simulated Reaction Times.

<table>
<thead>
<tr>
<th></th>
<th>Empirical RT</th>
<th></th>
<th>Synthetic RT</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>N</td>
<td>M</td>
<td>SD</td>
<td>N</td>
<td>SE</td>
</tr>
<tr>
<td>Congruent trials</td>
<td>1.39</td>
<td>0.29</td>
<td>202</td>
<td>1.38</td>
<td>0.27</td>
<td>155</td>
<td>0.03</td>
</tr>
<tr>
<td>Incongruent trials</td>
<td>1.56</td>
<td>0.31</td>
<td>200</td>
<td>1.61</td>
<td>0.37</td>
<td>152</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Note. The alpha level was set at 0.05. RT: Reaction time.

Finally, correlational analysis was used to check whether all EZ-DM parameter estimates (drift rate, boundary separation, and nondecision time) were associated with Flanker performance (see Table 7). Note that these correlations are for a subset of 150 children for whom there was available data. These analyses revealed that no EZ-DM parameters were statistically significantly associated with Flanker performance.

Table 7. Bivariate Correlations amongst EZ-DM, motor, and cognitive variables.

<table>
<thead>
<tr>
<th>Contextual variables</th>
<th>Motor control</th>
<th>ADHD symptoms</th>
<th>EZ-DM parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>IQ</td>
<td>TP</td>
<td>TR</td>
</tr>
<tr>
<td>IQ</td>
<td>-0.41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TP</td>
<td>-0.08</td>
<td>-0.07</td>
<td></td>
</tr>
<tr>
<td>TR</td>
<td>-0.03</td>
<td>0.2</td>
<td>0.07</td>
</tr>
<tr>
<td>PU</td>
<td>0.12</td>
<td>0.17</td>
<td>0.04</td>
</tr>
<tr>
<td>HI</td>
<td>0.19</td>
<td>-0.04</td>
<td>-0.11</td>
</tr>
<tr>
<td>DR</td>
<td>0.22</td>
<td>0.13</td>
<td>-0.05</td>
</tr>
<tr>
<td>BS</td>
<td>0.15</td>
<td>-0.06</td>
<td>-0.07</td>
</tr>
<tr>
<td>NDT</td>
<td>-0.45</td>
<td>0.17</td>
<td>0.1</td>
</tr>
<tr>
<td>FL</td>
<td>-0.3</td>
<td>0.37</td>
<td>-0.01</td>
</tr>
</tbody>
</table>

Appendix K. Assumption Checks for Multiple Linear Regression Analyses in Paper 2

As shown in Figures 9 – 15, the homogeneity of variance, low multicollinearity, normality of residuals, and lack of influential observations assumptions were roughly approximated in all multiple linear regression analyses.

Figure 9. Assumption Checks for Multiple Linear Regression of Flanker (Cognitive Inhibition) Performance.

Figure 10. Assumption Checks for Multiple Linear Regression of Backwards Digit Recall (Working Memory) Performance.
Figure 11. Assumption Checks for Multiple Linear Regression of Mister X (Working Memory) Performance.

Figure 12. Assumption Checks for Multiple Linear Regression of Mister X Processing (working Memory Manipulation) Performance.
Figure 13. Assumption Checks for Multiple Linear Regression of Dimensional Change Card Sort (Cognitive Flexibility) Performance.

Figure 14. Assumption Checks for Multiple Linear Regression of Drift Rate (Cognitive Modelling: Processing Efficiency).
Figure 15. Assumption checks for multiple linear regression of boundary separation (Cognitive Modelling: Speed-Accuracy Trade-off).

Figure 16. Assumption checks for multiple linear regression of nondecision time (Cognitive Modelling: Stimuli Encoding and Motor Response Preparation Time).
Appendix L: R Markdown Code and Output for Paper 2

Prepare R Studio

Clear R's brain

```r
rm(list = ls()) # "Resets" the R environment after any previous analysis, etc.
```

Prepare R Markdown

```r
options(scipen = 1, digits = 3)
```

Load packages

```r
library(haven) # For loading SPSS files (.sav)
library(readxl) # For loading .xlsx files
library(stringr) # For removing characters from data
library(data.table) # For renaming variables en masse
library(Hmisc) # For creating correlation matrix with p values
## Loading required package: lattice
## Loading required package: survival
## Loading required package: Formula
## Loading required package: ggplot2
```

```r
## Attaching package: 'Hmisc'
## The following objects are masked from 'package:base':
##   format.pval, units
library(huge) # For transforming data
library(interactions) # For post-hoc simple slope analysis
library(ggplot2) # For plotting
library(ggpubr) # For plotting
```

```r
## Registered S3 methods overwritten by 'broom':
##   method            from
##   tidy.glht         jtools
##   tidy.summary.glht jtools
library(tidyr) # For preparing data for plotting
library(gvlma) # For checking assumptions of mlr
library(performance) # For checking assumptions of mlr
library(see) # For plotting model asm
```

Part 1: Which elements of cognitive control are predicted by which parts of cognitive control?

Prepare data

Load data

```r
# Data from the Neurodevelopmental Assessment Unit database
# Includes demographic variables, symptom data, and age-corrected scores for the dependent
# variables for part 1 of the study
NDAU_data <- read_sav("NDAU_database_70.sav")
```

```r
# Data from the Amsterdam Neuropsychological Tasks Tapping Task
ANT_tapping_data_Z <- read.csv("ANT_Tapping_Zscores.csv")
```

```r
# Data from the Amsterdam Neuropsychological Tasks Tracking Task
ANT_tracking_data_Z <- read.csv("ANT_Tracking_Zscores.csv")
```

```r
# Data from the Amsterdam Neuropsychological Tasks Pursuit Task
ANT_pursuit_data_Z <- read.csv("ANT_Pursuit_Zscores.csv")
```

Select relevant variables

From NDAU database

```r
# Select demographic, symptom data, and age-corrected scores for the dependent variables for
# the NDAU database
NDAU_data <- NDAU_data[,c("ParticipantID", # Participant identification code which will be used to merge data sets
                           "Lucid_General_Standard", # Lucid Ability Test estimate of general cognitive ability for exclusion criteria
                           "Gender", "Age", # Demographic data
                           "hypImpScore", "inattDimScore", # DAWBA Hyperactive-Impulsive symptom score for moderation analysis
                           "hypImpScore", "inattDimScore", # DAWBA Inattentive symptom score to characteri
```
## Likely ADHD diagnosis for sample characteristics

The term "ADHD" refers to a neurodevelopmental disorder that typically presents in childhood and can persist into adulthood. It is characterized by a persistently patterns of inattention, hyperactivity, and impulsivity that cause significant distress or impairment in social, academic, or occupational functioning. The symptoms of ADHD can be divided into two main categories: inattention and hyperactivity-impulsivity.

- **Inattention** includes difficulties with sustained attention, task completion, and memory. Individuals with ADHD often have trouble focusing on tasks, may daydream, and may have difficulty staying organized.
- **Hyperactivity-impulsivity** involves excess movements, fidgeting, and inability to remain seated. Individuals with ADHD may have difficulty waiting their turn, may blurt out answers, and may have trouble controlling their behavior in a manner appropriate for their age.

ADHD is often diagnosed based on the presence of these symptoms along with severity and impact on daily functioning. Diagnosis requires the symptoms to be present for at least six months and to cause significant impairment in multiple areas of life, such as academic, social, or occupational settings.
# Rename variables

```r
```

Exclusion criteria

IQ

# Exclude participants with IQ < 70
```r
part1_data <- part1_data[part1_data$IQ >= 70, ]
```

Data exploration and cleansing

Demographic variables

# Label genders
```r
part1_data$Gender <- factor(part1_data$Gender, levels = c(1,2), labels = c("Male","Female")
```

# Create vector of demographic variables
```r
part1_demographics <- c("Age", "IQ"
```

# Convert age in months to age in years
```r
part1_data$Age <- part1_data$Age / 12
```

# Create histograms for all continuous demographic variables
```r
ggplot(gather(subset(part1_data[,c(part1_demographics)]), aes(value))) + geom_histogram(bins = 15, fill = "#0c4c8a") + facet_wrap(~key, scales = 'free_x')
```

## Warning: Removed 27 rows containing non-finite values (stat_bin).
# Calculate descriptive statistics for all continuous demographic variables
summary(subset(part1_data[c("Age","IQ")]), text = TRUE)
##       Age             IQ
##  Min.   :4.25   Min.   : 74.0
##  1st Qu.:5.67   1st Qu.: 90.0
##  Median :6.50   Median : 98.0
##  Mean   :6.49   Mean   : 98.3
##  3rd Qu.:7.42   3rd Qu.:105.0
##  Max.   :9.75   Max.   :137.0
##  NA's     :16     NA's   :11

# Calculate standard deviations for all continuous demographic variables
round(sapply(subset(part1_data[c("Age","IQ")]), sd, na.rm = TRUE), 2)
##   Age    IQ
##  1.05 11.96

# Calculate median absolute deviations for all continuous demographic variables
round(sapply(subset(part1_data[c("Age","IQ")]), mad, na.rm = TRUE), 2)
##   Age    IQ
##  1.36 11.86

# Count number of males and females
sum(part1_data$Gender == 'Male', na.rm = TRUE)
## [1] 164
sum(part1_data$Gender == 'Female', na.rm = TRUE)
## [1] 78

Predictor variables
# Recode missing value signifiers as NAs
# Hyperactive-impulsive symptoms
part1_data$HypImp[part1_data$HypImp == 99] <- NA
# Create vector of predictor variables
part1_predictors <- c("Tapping",
                      "Tracking",
                      "Tracking SD",
                      "Pursuit",
                      "Pursuit SD",
                      "Age",
                      "IQ")


# Re-sign predictor variables so that higher scores reflect better performance

# ANT Tracking task data
# Mean deviation from ideal trajectory
part1_data$Tracking <- part1_data$Tracking * -1  # Now higher scores = better

# Standard deviation of mean deviation from ideal trajectory
part1_data$Tracking SD` <- part1_data$Tracking SD` * -1

# ANT Pursuit task data
# Mean deviation from ideal trajectory
part1_data$Pursuit <- part1_data$Pursuit * -1

# Standard deviation of mean deviation from ideal trajectory
part1_data$Pursuit SD` <- part1_data$Pursuit SD` * -1

# Create histograms for all predictor variables
ggplot(gather(subset(part1_data[c(part1_predictors)]), aes(value)) + geom_histogram(bins = 15, fill = "#0c4c8a") + facet_wrap(~key, scales = 'free_x')
## Warning: Removed 169 rows containing non-finite values (stat_bin).

# Calculate descriptive statistics for all predictor variables
summary(subset(part1_data[c(part1_predictors)]), text = T)
##
# Tapping     Tracking     Tracking SD   Pursuit
# Min. : -7.02  Min. : -22.46  Min. : -16.23 Min. : -63.0
# 1st Qu.: -1.99 1st Qu.: -3.27 1st Qu.: -1.24 1st Qu.: -2.5
# Median:  0.38 Median:  -1.40 Median:  -0.30 Median:  -0.7
# Mean :  0.84 Mean :  -2.37 Mean :  -0.88 Mean :  -2.1
# 3rd Qu.:  2.50 3rd Qu.:  -0.18 3rd Qu.:  0.00 3rd Qu.:  0.1
# Max. :  2.50 Max. :  0.61 Max. :  0.95 Max. :  1.6
# NA’s :11 NA’s :11 NA’s :11 NA’s :11
# Calculate standard deviations
round(sapply(subset(part1_data[,c(part1_predictors)]), sd, na.rm = TRUE), 2)
##
##     Tapping    Tracking Tracking SD     Pursuit  Pursuit SD      HypImp
##        1.72        3.26        1.90        5.35        3.81        6.20
##       InAtt
##        5.98

# Calculate median absolute deviations
round(sapply(subset(part1_data[,c(part1_predictors)]), mad, na.rm = TRUE), 2)
##
##     Tapping    Tracking Tracking SD     Pursuit  Pursuit SD      HypImp
##        1.06        2.07        0.67        1.66        1.65        6.67
##       InAtt
##        5.93

# Create correlation matrix for predictor variables
round(cor(subset(part1_data[,c(part1_predictors)]), use = "pairwise.complete.obs"), 2)
##
##         Tapping Tracking Tracking SD Pursuit Pursuit SD HypImp InAtt
## Tapping        1.00     0.08        0.07     -0.11 -0.10 -0.08 -0.04
## Tracking      0.08     1.00        0.91      0.18  0.22  0.03 -0.07
## Tracking SD   0.07     0.91        1.00      0.16  0.20 -0.06 -0.10
## Pursuit      -0.11    -0.18       -0.16     1.00  0.90  0.01 -0.02
## Pursuit SD   -0.10    -0.22       -0.20     0.90  1.00 -0.02 -0.04
## HypImp       -0.08    -0.03      -0.06      0.01 -0.02 1.00  0.86
## InAtt        -0.04    -0.07      -0.10     -0.02 -0.04 0.86  1.00

Dependent variables

# Create a vector of dependent variables
part1_dependents <- c("Flanker", "DCCS", "BDR", "MRX", "MRXproc")

# Replace missing values with NAs
# For NIH Flanker
part1_data$Flanker[part1_data$Flanker == 999] <- NA

# For NIH DCCSs
part1_data$DCCS[part1_data$DCCS == 999] <- NA

# For AWMA BDR
part1_data$BDR[part1_data$BDR == 999] <- NA

# For AWMA Mr X
part1_data$MRX[part1_data$MRX == 999] <- NA
part1_data$MRXproc[part1_data$MRXproc == 999] <- NA

# Create histograms for all dependent variables
ggplot(gather(subset(part1_data[,c(part1_dependents)])), aes(value)) +
  geom_histogram(bins = 15, fill = "#0c4c8a") +
  facet_wrap(~key, scales = "free_x")

# Warning: attributes are not identical across measure variables;
# they will be dropped
# Warning: Removed 292 rows containing non-finite values (stat_bin).
# Calculate descriptive statistics for all dependent variables
summary(subset(part1_data[c(part1_dependents)], text = T))
##     Flanker         DCCS            BDR             MRX         MRXproc
##  Min.   : 54   Min.   : 41.0   Min.   : 64.0   Min.   : 71   Min.   : 78
##  1st Qu.: 83   1st Qu.: 87.0   1st Qu.: 87.0   1st Qu.: 94   1st Qu.: 92
##  Median : 94   Median : 96.0   Median : 98.0   Median :104   Median : 99
##  Mean   : 93   Mean   : 94.5   Mean   : 98.7   Mean   :106   Mean   :104
##  3rd Qu.:102   3rd Qu.:100.0   3rd Qu.:108.5   3rd Qu.:116   3rd Qu.:120
##  Max.   :131   Max.   :146.0   Max.   :150.0   Max.   :148   Max.   :160
##  NA's   :59    NA's   :40      NA's   :44      NA's   :77    NA's   :72

# Calculate standard deviations
round(sapply(subset(part1_data[c(part1_dependents)]), sd, na.rm = TRUE), 2)
## Flanker    DCCS     BDR     MRX MRXproc
##    14.9    14.1    16.4    17.2    16.6

# Calculate median absolute deviations
round(sapply(subset(part1_data[c(part1_dependents)]), mad, na.rm = TRUE), 2)
## Flanker    DCCS     BDR     MRX MRXproc
##    11.9    10.4    16.3    17.8    13.3

# Create correlation matrix for dependent variables
round(cor(subset(part1_data[c(part1_dependents)]), use = "pairwise.complete.obs"), 2)
##         Flanker DCCS  BDR  MRX MRXproc
## Flanker    1.00 0.32 0.41 0.36    0.40
## DCCS       0.32 1.00 0.33 0.22    0.27
## BDR        0.41 0.33 1.00 0.33    0.43
## MRX        0.36 0.22 0.33 1.00    0.87
## MRXproc    0.40 0.27 0.43 0.87    1.00

Outlier removal
# Pre transformation histograms
part1_data[c("Age","IQ","Tapping","Tracking","Pursuit","Flanker","BDR","MRX","MRXproc","DCCS")]
  %>%
gather() %>%
ggplot(aes(value)) +
```r
facet_wrap(~ key, scales = "free_x") +
geom_histogram(bins = 10, fill = "#0c4c8a")

## Warning: attributes are not identical across measure variables;
## they will be dropped
## Warning: Removed 352 rows containing non-finite values (stat_bin).

# Remove outliers from...
# Pursuit
part1_data$Pursuit[part1_data$Pursuit == -62.98] <- NA

# Tracking
part1_data$Tracking[part1_data$Tracking == -22.46] <- NA
part1_data$Tracking[part1_data$Tracking == -22.43] <- NA

Transformation
# Nonparanormal transformation
# Specify data for transformation
trans_data <- subset(part1_data[c(part1_demographics, # Demographic variables (e.g., age & IQ)
part1_predictors, # Predictor variables (e.g., ANT tasks)
part1_dependents # Dependent variables (e.g, Flanker) ]))

# Perform transformation
trans_data <- huge.npn(trans_data) # Runs transformation
## Conducting the nonparanormal (nnp) transformation via shrunkun ECDF....done.
trans_data <- as.data.frame(trans_data) # Ensures output is recognised as data frame by R

# Save transformed data to file
write.csv(trans_data, "transformed_data.csv")
# Post transformation histograms
trans_data[c("Age","IQ","Tapping","Tracking","Pursuit","Flanker","BDR","MRX","MRXproc","DCCS")]
%>%
gather() %>%
```
ggplot(aes(value)) +
  facet_wrap(~ key, scales = "free_x") +
  geom_histogram(bins = 10, fill = "#0c4c8a")

# Remove all objects apart from the compiled data frame for Part 1 of the study
rm(list = ls()[-ls()%in%c("part1_data", "trans_data")])

# Prevents my laptop from being overwhelmed

Correlational analyses
# Make correlation matrix
round(cor(trans_data, use = "pairwise.complete.obs", method = "pearson"), 2)

# Only display significant (< 0.05) values
ifelse(corr_p_matrix < 0.05, corr_p_matrix, "NS")
## Tracking SD "NS" "NS" "0" "0.001" "NS" "NS" "NS"
## Pursuit "NS" "NS" "0" "0.003" "0.001" "NS" "NS"
## Pursuit SD "NS" "NS" "0" "0.014" "0" "NS" "NS"
## HypImp NA "0" "NS" "NS" "0" "0" "0"
## InAtt "0" NA "NS" "NS" "0" "0" "0"
## Flanker "NS" "NS" NA "0" "0.028" "NS" "NS"
## DCCS "NS" "NS" "0" NA "0.028" "NS" "NS"
## BDR "NS" "NS" NA "0" "0" "0" "0"
## MRX "0" "0" "NS" "NS" "0" NA "0"
## MRXproc "0" "0" "NS" "NS" "0" "0" NA

# Calculate 95% CIs for key correlations for write up
# For Tracking and Flanker
cor.test(trans_data$Tracking, trans_data$Flanker, method = "pearson")
##
##  Pearson's product-moment correlation
##
## data:  trans_data$Tracking and trans_data$Flanker
## t = 4, df = 253, p-value = 0.0003
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  0.105 0.339
## sample estimates:
##   cor
## 0.225

# For Tracking and DCCS
cor.test(trans_data$Tracking, trans_data$DCCS, method = "pearson")
##
##  Pearson's product-moment correlation
##
## data:  trans_data$Tracking and trans_data$DCCS
## t = 2, df = 253, p-value = 0.02
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  0.0197 0.2605
## sample estimates:
##   cor
## 0.142

# For Tracking and hyperactive-impulsive symptoms
cor.test(trans_data$Tracking, trans_data$HypImp, method = "pearson")
##
##  Pearson's product-moment correlation
##
## data:  trans_data$Tracking and trans_data$HypImp
## t = 2, df = 253, p-value = 0.04
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  0.00432 0.24610
## sample estimates:
##   cor
## 0.127

# For Pursuit and Flanker
cor.test(trans_data$Pursuit, trans_data$Flanker, method = "pearson")
##
##  Pearson's product-moment correlation
##
## data:  trans_data$Pursuit and trans_data$Flanker
## t = 4, df = 253, p-value = 1e-05
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  0.153 0.381
## sample estimates:
##   cor
## 0.271

# For Pursuit and DCCS
cor.test(trans_data$Pursuit, trans_data$DCCS, method = "pearson")
##
##  Pearson's product-moment correlation
##
## data:  trans_data$Pursuit and trans_data$DCCS
## t = 2, df = 253, p-value = 0.02
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  0.0197 0.2605
## sample estimates:
##   cor
## 0.142
## Pearson's product-moment correlation

### data: trans_data$Pursuit and trans_data$DCCS
### t = 3, df = 253, p-value = 0.003
### alternative hypothesis: true correlation is not equal to 0
### 95 percent confidence interval:
### 0.0666 0.3037
### sample estimates:
### cor
### 0.188

# For Pursuit and BDR
cor.test(trans_data$Pursuit, trans_data$BDR, method = "pearson")

### Pearson's product-moment correlation
### data: trans_data$Pursuit and trans_data$BDR
### t = 3, df = 253, p-value = 0.0007
### alternative hypothesis: true correlation is not equal to 0
### 95 percent confidence interval:
### 0.0896 0.3246
### sample estimates:
### cor
### 0.21

# For hyperactive-impulsive symptoms and Mr X
cor.test(trans_data$HypImp, trans_data$MRX, method = "pearson")

### Pearson's product-moment correlation
### data: trans_data$HypImp and trans_data$MRX
### t = 6, df = 253, p-value = 1e-09
### alternative hypothesis: true correlation is not equal to 0
### 95 percent confidence interval:
### 0.260 0.472
### sample estimates:
### cor
### 0.371

# For hyperactive-impulsive symptoms and Mr X processing
cor.test(trans_data$HypImp, trans_data$MRXproc, method = "pearson")

### Pearson's product-moment correlation
### data: trans_data$HypImp and trans_data$MRXproc
### t = 6, df = 253, p-value = 4e-09
### alternative hypothesis: true correlation is not equal to 0
### 95 percent confidence interval:
### 0.245 0.460
### sample estimates:
### cor
### 0.357

### Inferential analyses

#### Inhibition

# Perform multiple linear regression
part1_inhibition_mlr <- lm(Flanker ~ Tapping * HypImp + Tracking, 
data=trans_data, na.action = na.omit)

# Ensure assumptions for linear regression are met before inspecting results
check_model(part1_inhibition_mlr)

# View results
summary(part1_inhibition_mlr)

### Call:
### lm(formula = Flanker ~ Tapping * HypImp + Tracking, data = trans_data, 
###    na.action = na.omit)
# Residuals:
##     Min      1Q  Median      3Q     Max
##-2.5697  -0.6632 -0.0014  0.6704  2.6018

# Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)  0.000812    0.060898    0.01    0.989
## Tapping     -0.070362    0.066049   -1.07    0.288
## HypImp       0.095656    0.063028    1.52    0.130
## Tracking    0.214460    0.064591    3.32  0.001 **
## Tapping:HypImp 0.077856    0.074049   -1.05    0.294

---
# Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

# Residual standard error: 0.972 on 250 degrees of freedom
# Multiple R-squared:  0.0657, Adjusted R-squared:  0.0508
# F-statistic: 4.4 on 4 and 250 DF,  p-value: 0.00188

# View terms p values to 3 decimal places
print(inhibition_mlr_pvals <- round(summary(part1_inhibition_mlr)$coefficients[,4], 3))

##    (Intercept)        Tapping         HypImp Tracking Tapping:HypImp
##          0.989          0.288          0.130          0.001          0.294

# View 95% CIs for significant terms
round(confint(part1_inhibition_mlr, 'Tracking', level=0.95), 2)

##          2.5 % 97.5 %
## Tracking  0.09   0.34

Working memory

Verbal
# Perform multiple linear regression
part1_verbalWM_mlr <- lm(BDR ~ Tapping * HypImp,
                        data=trans_data, na.action = na.omit)

# Examine assumptions
check_model(part1_verbalWM_mlr)

# View results
summary(part1_verbalWM_mlr)

## Call:
## lm(formula = BDR ~ Tapping * HypImp, data = trans_data, na.action = na.omit)
## Residuals:
##     Min      1Q  Median      3Q     Max
##-2.4869  -0.6780  0.0045  0.6682  2.7106

## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)  0.00276    0.06213    0.04    0.966
## Tapping     -0.07757    0.06620   -1.17    0.247
## HypImp       0.00822    0.06368    0.13    0.897
## Tapping:HypImp 0.12004    0.07444    1.61    0.108

## Residual standard error: 0.992 on 251 degrees of freedom
## Multiple R-squared:  0.0223, Adjusted R-squared:  0.0106
## F-statistic: 1.91 on 3 and 251 DF,  p-value: 0.129

# View terms p values to 3 decimal places
print(verbalWM_mlr_pvals <- round(summary(part1_verbalWM_mlr)$coefficients[,4], 3))

##    (Intercept)        Tapping         HypImp Tapping:HypImp
##          0.965          0.242          0.970          0.108

Visuospatial

Foundational
# Perform multiple linear regression
part1_visspWM_mlr <- lm(MRX ~ Tapping * HypImp,
# Examine assumptions
ccheck_model(part1_visspWM_mlr)

# View results
summary(part1_visspWM_mlr)

## Call:
## lm(formula = MRX ~ Tapping * HypImp, data = trans_data, na.action = na.omit)
##
## Residuals:
##    Min     1Q Median     3Q    Max
##-2.276  -0.594  0.119  0.650  2.977
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   -0.0018     0.0582  -0.03    0.98
## Tapping       0.0660     0.0620   1.06    0.29
## HypImp        0.3777     0.0596   6.34  1.1e-09 ***
## Tapping:HypImp -0.0513     0.0697  -0.74    0.46
## ---
## Signif. codes:  *  **  ***   0.05 0.01 0.001 1
##
## Residual standard error: 0.928 on 251 degrees of freedom
## Multiple R-squared: 0.142, Adjusted R-squared: 0.132
## F-statistic: 13.9 on 3 and 251 DF,  p-value: 2.16e-08

# View terms p values to 3 decimal places
print(visspWM_mlr_pvals <- round(summary(part1_visspWM_mlr)$coefficients[,4], 3))

## (Intercept)  Tapping  HypImp  Tapping:HypImp
## 0.975        0.288    0.000    0.462

# View 95% CIs for significant terms
round(confint(part1_visspWM_mlr, 'HypImp', level=0.95), 2)

## 2.5 97.5 %
## HypImp  0.26 0.5

# Perform multiple linear regression
part1_visspWMproc_mlr <- lm(MRXproc ~ Tracking * HypImp + Pursuit * HypImp, 
       data=trans_data, na.action = na.omit)

# Examine assumptions
ccheck_model(part1_visspWMproc_mlr)

# View results
summary(part1_visspWMproc_mlr)

## Call:
## lm(formula = MRXproc ~ Tracking * HypImp + Pursuit * HypImp, 
##     data = trans_data, na.action = na.omit)
##
## Residuals:
##    Min     1Q Median     3Q    Max
##-2.8862  -0.6471  0.0942  0.6663  2.9799
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)    -0.0165     0.0592  -0.28    0.78
## Tracking      -0.0396     0.0695  -0.57    0.57
## HypImp         0.3574     0.0608   5.87  1.4e-08 ***
## Pursuit        0.0597     0.0691   0.86    0.39
## Tracking:HypImp 0.0759     0.0788   0.96    0.34
## HypImp:Pursuit  0.0465     0.0765   0.61    0.54
## ---
## Signif. codes:  *  **  ***   0.05 0.01 0.001 1
##
## Residual standard error: 0.972 on 250 degrees of freedom
## Multiple R-squared: 0.148, Adjusted R-squared: 0.136
## F-statistic: 14.3 on 6 and 250 DF,  p-value: 1.66e-08
## Residual standard error: 0.935 on 249 degrees of freedom
## Multiple R-squared:  0.14,  Adjusted R-squared:  0.123
## F-statistic: 8.14 on 5 and 249 DF,  p-value: 3.93e-07

### View terms p values to 3 decimal places

```
print(visspWMproc_mlr_pvals <- round(summary(part1_visspWMproc_mlr)$coefficients[,4], 3))
```

### (Intercept) Tracking HypImp Pursuit Tracking:HypImp
### 0.781 0.569 0.000 0.389 0.336

### HypImp:Pursuit
### 0.544

### View 95% CIs for significant terms

```
round(confint(part1_visspWM_mlr, 'HypImp', level=0.95), 2)
```

### 2.5 % 97.5 %

### HypImp 0.26 0.5

### Switching/flexibility

### Perform multiple linear regression

```
part1_switching_mlr <- lm(DCCS ~ Tapping * HypImp + Tracking + Pursuit, data = trans_data, na.action = na.omit)
```

### Examine assumptions

```
check_model(part1_switching_mlr)
```

### View results

```
summary(part1_switching_mlr)
```

### Call:

```
lm(formula = DCCS ~ Tapping * HypImp + Tracking + Pursuit, data = trans_data, na.action = na.omit)
```

### Residuals:

```
     Min      1Q  Median      3Q     Max
-2.6000 -0.6491  0.0788  0.6049  2.7245
```

### Coefficients:

```
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.000412   0.061573   0.01  0.995
Tapping      0.092213   0.066795   1.38  0.169
HypImp       0.060252   0.063847   0.94  0.346
Tracking     0.035590   0.074507   0.48  0.633
Pursuit      0.152136   0.072653   2.09  0.037 *
Tapping:HypImp -0.009864   0.075350  -0.13  0.896
```

### Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

### Residual standard error: 0.983 on 249 degrees of freedom
### Multiple R-squared:  0.0486, Adjusted R-squared:  0.0295
### F-statistic: 2.55 on 5 and 249 DF,  p-value: 0.0287

### View terms p values to 3 decimal places

```
print(switching_mlr_pvals <- round(summary(part1_switching_mlr)$coefficients[,4], 3))
```

### (Intercept) Tapping HypImp Tracking Pursuit
### 0.995 0.169 0.346 0.633 0.037

### Tapping:HypImp
### 0.896

### View 95% CIs for significant terms

```
round(confint(part1_switching_mlr, 'Pursuit', level=0.95), 2)
```

### 2.5 % 97.5 %

### Pursuit 0.01 0.3
Part 2: Which processes contributing to flanker performance are predicted by motor generation and/or visuomotor control?

Prepare data

Load data

```r
# Load
data part2_EZ_cong <- read_excel("EZDM_empirical_congruent.xlsx")
data part2_EZ_incong <- read_excel("EZDM_empirical_incongruent.xlsx")
```

# Select variables

```r
# Select variables
data part2_EZ_cong <- part2_EZ_cong[, c("Participant", "driftRate", "boundSep", "nondecTime", "trialNum")]
data part2_EZ_incong <- part2_EZ_incong[, c("Participant", "driftRate", "boundSep", "nondecTime", "trialNum")]
```

# Rename

```r
# Rename
setnames(part2_EZ_cong, old = c("driftRate", "boundSep", "nondecTime", "trialNum"),
          new = c("CONGdriftRate", "CONGboundSep", "CONGnondecTime", "CONGtrialNum"))
setnames(part2_EZ_incong, old = c("driftRate", "boundSep", "nondecTime", "trialNum"),
          new = c("INCONGdriftRate", "INCONGboundSep", "INCONGnondecTime", "INCONGtrialNum"))
```

Add data for Part 2 to data for Part 1

# Merge

```r
# Merge
part2_data <- merge(part2_data, part2_EZ_cong, by.x = "Participant", by.y = "Participant")
part2_data <- merge(part2_data, part2_EZ_incong, by.x = "Participant", by.y = "Participant")
```

# Sort data frame by participant ID (Lowest first)

```r
# Sort data frame by participant ID (Lowest first)
part2_data <- part2_data[order(as.numeric(part2_data$Participant)),]
```

Exclusion

Exclude participants with negative NDT estimates as these denote poor model fit

```r
# Exclude participants with negative NDT estimates arising from congruent trials
part2_data <- part2_data[part2_data$CONGnondecTime > 0,]
```

```r
# Exclude participants with negative NDT estimates arising from incongruent trials
part2_data <- part2_data[part2_data$INCONGnondecTime > 0,]
```

Exclude participants with no DDM parameter estimates due to poor accuracy

```r
# Exclude participants with no EZ-DM parameter estimates for congruent trials as a result of low accuracy (50% or less)
```
part2_data <- part2_data[!is.na(part2_data$CONGdriftRate),]

# Exclude participants with no EZ-DM parameter estimates for incongruent trials as a result of low accuracy (50% or less)
part2_data <- part2_data[!is.na(part2_data$INCONGdriftRate),]

Calculate dependent variables which are a combination of EZ-DM estimates for congruent and incongruent trials

# Combined (congruent & incongruent) boundary separation estimates
part2_data$COMBINEDboundSep <- (as.numeric(part2_data$CONGboundSep) + as.numeric(part2_data$INCONGboundSep))/2

# Combined (congruent & incongruent) nondecision time estimates
part2_data$COMBINEDnondecTime <- (as.numeric(part2_data$CONGnondecTime) + as.numeric(part2_data$INCONGnondecTime))/2

Check robustness of EZ-DM parameter estimates Use construct-samples from the fast-DM software to perform parameter recovery (relative accuracy)

# Renumber participant IDs for construct-samples to work smoothly
part2_data$newRID <- c(1:nrow(part2_data)) # New IDs starting from 1

# Extract empirical DDM parameter estimates & paste into construct-samples format
# For congruent trials
# Prepare list of commands to run construct samples
cong_list_run <- paste0("construct-samples.exe", "-a", round((part2_data$CONGboundSep * 10), 2), "-v", round(part2_data$CONGdriftRate * 10, 2), "-t", round(part2_data$CONGnondecTime, 2), "-N 1 -n 24 -p 3 -r -o ") # Specifies number of subjects, number of trials, precision of estimation, and randomisation (true)

cong_list_run <- paste0(cong_list_run, "temp", part2_data$newRID, ".lst")

# Prepare list of participant numbers to store results
cong_list_store <- as.character(paste0("temp", part2_data$newRID, ",.lst"))

# For incongruent trials
# Prepare list of commands to run construct samples
incong_list_run <- paste0("construct-samples.exe", "-a", round((part2_data$INCONGboundSep * 10), 2), "-v", round(part2_data$INCONGdriftRate * 10, 2), "-t", round(part2_data$INCONGnondecTime, 2), "-N 1 -n 16 -p 3 -r -o ")

incong_list_run <- paste0(incong_list_run, "temp", part2_data$newRID, ",.lst")

# Prepare list of participant numbers to store results
incong_list_store <- as.character(paste0("temp", part2_data$newRID, ",.lst"))

For congruent trials
# Run construct-samples
# For congruent trials
for (sub in 1:1)
  system(cong_list_run[1]))
  system(cong_list_run[2]))
  system(cong_list_run[3]))
  system(cong_list_run[4]))
  system(cong_list_run[5]))
  system(cong_list_run[6])]}
system(cong_list_run[[66]])
system(cong_list_run[[67]])
system(cong_list_run[[68]])
system(cong_list_run[[69]])
system(cong_list_run[[70]])

system(cong_list_run[[71]])
system(cong_list_run[[72]])
system(cong_list_run[[73]])
system(cong_list_run[[74]])
system(cong_list_run[[75]])
system(cong_list_run[[76]])
system(cong_list_run[[77]])
system(cong_list_run[[78]])
system(cong_list_run[[79]])
system(cong_list_run[[80]])

system(cong_list_run[[81]])
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system(cong_list_run[[88]])
system(cong_list_run[[89]])
system(cong_list_run[[90]])

system(cong_list_run[[91]])
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system(cong_list_run[[100]])

system(cong_list_run[[101]])
system(cong_list_run[[102]])
system(cong_list_run[[103]])
system(cong_list_run[[104]])
system(cong_list_run[[105]])
system(cong_list_run[[106]])
system(cong_list_run[[107]])
system(cong_list_run[[108]])
system(cong_list_run[[109]])
system(cong_list_run[[110]])

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system(cong_list_run[[118]])
system(cong_list_run[[119]])
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system(cong_list_run[[121]])
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system(cong_list_run[[123]])
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system(cong_list_run[[126]])
system(cong_list_run[[127]])
system(cong_list_run[[128]])
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system(cong_list_run[[131]])
system(cong_list_run[[132]])
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system(cong_list_run[[141]])
system(cong_list_run[[142]])
system(cong_list_run[[143]])
system(cong_list_run[[144]])
system(cong_list_run[[145]])
system(cong_list_run[[146]])
system(cong_list_run[[147]])
system(cong_list_run[[148]])
system(cong_list_run[[149]])

S1 = read.table(cong_list_store[[1]], header = FALSE)
S2 = read.table(cong_list_store[[2]], header = FALSE)
S3 = read.table(cong_list_store[[3]], header = FALSE)
S4 = read.table(cong_list_store[[4]], header = FALSE)
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S85 = read.table(cong_list_store[[85]], header = FALSE)
S86 = read.table(cong_list_store[[86]], header = FALSE)
S87 = read.table(cong_list_store[[87]], header = FALSE)
S188 = read.table(cong_list_store[[88]], header = FALSE)
S189 = read.table(cong_list_store[[89]], header = FALSE)
S190 = read.table(cong_list_store[[90]], header = FALSE)

S191 = read.table(cong_list_store[[91]], header = FALSE)
S192 = read.table(cong_list_store[[92]], header = FALSE)
S193 = read.table(cong_list_store[[93]], header = FALSE)
S194 = read.table(cong_list_store[[94]], header = FALSE)
S195 = read.table(cong_list_store[[95]], header = FALSE)
S196 = read.table(cong_list_store[[96]], header = FALSE)
S197 = read.table(cong_list_store[[97]], header = FALSE)
S198 = read.table(cong_list_store[[98]], header = FALSE)
S199 = read.table(cong_list_store[[99]], header = FALSE)
S200 = read.table(cong_list_store[[100]], header = FALSE)

S201 = read.table(cong_list_store[[101]], header = FALSE)
S202 = read.table(cong_list_store[[102]], header = FALSE)
S203 = read.table(cong_list_store[[103]], header = FALSE)
S204 = read.table(cong_list_store[[104]], header = FALSE)
S205 = read.table(cong_list_store[[105]], header = FALSE)
S206 = read.table(cong_list_store[[106]], header = FALSE)
S207 = read.table(cong_list_store[[107]], header = FALSE)
S208 = read.table(cong_list_store[[108]], header = FALSE)
S209 = read.table(cong_list_store[[109]], header = FALSE)
S210 = read.table(cong_list_store[[110]], header = FALSE)

S211 = read.table(cong_list_store[[111]], header = FALSE)
S212 = read.table(cong_list_store[[112]], header = FALSE)
S213 = read.table(cong_list_store[[113]], header = FALSE)
S214 = read.table(cong_list_store[[114]], header = FALSE)
S215 = read.table(cong_list_store[[115]], header = FALSE)
S216 = read.table(cong_list_store[[116]], header = FALSE)
S217 = read.table(cong_list_store[[117]], header = FALSE)
S218 = read.table(cong_list_store[[118]], header = FALSE)
S219 = read.table(cong_list_store[[119]], header = FALSE)
S220 = read.table(cong_list_store[[120]], header = FALSE)

S221 = read.table(cong_list_store[[121]], header = FALSE)
S222 = read.table(cong_list_store[[122]], header = FALSE)
S223 = read.table(cong_list_store[[123]], header = FALSE)
S224 = read.table(cong_list_store[[124]], header = FALSE)
S225 = read.table(cong_list_store[[125]], header = FALSE)
S226 = read.table(cong_list_store[[126]], header = FALSE)
S227 = read.table(cong_list_store[[127]], header = FALSE)
S228 = read.table(cong_list_store[[128]], header = FALSE)
S229 = read.table(cong_list_store[[129]], header = FALSE)
S230 = read.table(cong_list_store[[130]], header = FALSE)

S231 = read.table(cong_list_store[[131]], header = FALSE)
S232 = read.table(cong_list_store[[132]], header = FALSE)
S233 = read.table(cong_list_store[[133]], header = FALSE)
S234 = read.table(cong_list_store[[134]], header = FALSE)
S235 = read.table(cong_list_store[[135]], header = FALSE)
S236 = read.table(cong_list_store[[136]], header = FALSE)
S237 = read.table(cong_list_store[[137]], header = FALSE)
S238 = read.table(cong_list_store[[138]], header = FALSE)
S239 = read.table(cong_list_store[[139]], header = FALSE)
S240 = read.table(cong_list_store[[140]], header = FALSE)

S241 = read.table(cong_list_store[[141]], header = FALSE)
S242 = read.table(cong_list_store[[142]], header = FALSE)
S243 = read.table(cong_list_store[[143]], header = FALSE)
S244 = read.table(cong_list_store[[144]], header = FALSE)
S245 = read.table(cong_list_store[[145]], header = FALSE)
S246 = read.table(cong_list_store[[146]], header = FALSE)
```
S147 = read.table(cong_list_store[[147]], header = FALSE)
S148 = read.table(cong_list_store[[148]], header = FALSE)
S149 = read.table(cong_list_store[[149]], header = FALSE)
S150 = read.table(cong_list_store[[150]], header = FALSE)
S151 = read.table(cong_list_store[[151]], header = FALSE)
S152 = read.table(cong_list_store[[152]], header = FALSE)
S153 = read.table(cong_list_store[[153]], header = FALSE)
S154 = read.table(cong_list_store[[154]], header = FALSE)
S155 = read.table(cong_list_store[[155]], header = FALSE)

S = rbind(S11, S12, S13, S14, S15, S16, S17, S18, S19, S20,
S21, S22, S23, S24, S25, S26, S27, S28, S29, S30,
S31, S32, S33, S34, S35, S36, S37, S38, S39, S40,
S41, S42, S43, S44, S45, S46, S47, S48, S49, S50,
S51, S52, S53, S54, S55, S56, S57, S58, S59, S60,
S61, S62, S63, S64, S65, S66, S67, S68, S69, S70,
S71, S72, S73, S74, S75, S76, S77, S78, S79, S80,
S81, S82, S83, S84, S85, S86, S87, S88, S89, S90,
S91, S92, S93, S94, S95, S96, S97, S98, S99, S100,
S101, S102, S103, S104, S105, S106, S107, S108, S109, S110,
S111, S112, S113, S114, S115, S116, S117, S118, S119, S120,
S121, S122, S123, S124, S125, S126, S127, S128, S129, S130,
S131, S132, S133, S134, S135, S136, S137, S138, S139, S140,
S141, S142, S143, S144, S145, S146, S147, S148, S149, S150,
S151, S152, S153, S154, S155)

C = c(rep(1, 24), rep(2, 24), rep(3, 24), rep(4, 24), rep(5, 24), rep(6, 24), rep(7, 24), rep(8, 24), rep(9, 24), rep(10, 24),
rep(11, 24), rep(12, 24), rep(13, 24), rep(14, 24), rep(15, 24), rep(16, 24), rep(17, 24),
rep(18, 24), rep(19, 24), rep(20, 24),
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rep(28, 24), rep(29, 24), rep(30, 24),
rep(31, 24), rep(32, 24), rep(33, 24), rep(34, 24), rep(35, 24), rep(36, 24), rep(37, 24),
rep(38, 24), rep(39, 24), rep(40, 24),
rep(41, 24), rep(42, 24), rep(43, 24), rep(44, 24), rep(45, 24), rep(46, 24), rep(47, 24),
rep(48, 24), rep(49, 24), rep(50, 24),
rep(51, 24), rep(52, 24), rep(53, 24), rep(54, 24), rep(55, 24), rep(56, 24), rep(57, 24),
rep(58, 24), rep(59, 24), rep(60, 24),
rep(61, 24), rep(62, 24), rep(63, 24), rep(64, 24), rep(65, 24), rep(66, 24), rep(67, 24),
rep(68, 24), rep(69, 24), rep(70, 24),
rep(71, 24), rep(72, 24), rep(73, 24), rep(74, 24), rep(75, 24), rep(76, 24), rep(77, 24),
rep(78, 24), rep(79, 24), rep(80, 24),
rep(81, 24), rep(82, 24), rep(83, 24), rep(84, 24), rep(85, 24), rep(86, 24), rep(87, 24),
rep(88, 24), rep(89, 24), rep(90, 24),
rep(91, 24), rep(92, 24), rep(93, 24), rep(94, 24), rep(95, 24), rep(96, 24), rep(97, 24),
rep(98, 24), rep(99, 24), rep(100, 24),
rep(101, 24), rep(102, 24), rep(103, 24), rep(104, 24), rep(105, 24), rep(106, 24), rep(107, 24),
rep(108, 24), rep(109, 24), rep(110, 24),
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rep(138, 24), rep(139, 24), rep(140, 24),
rep(141, 24), rep(142, 24), rep(143, 24), rep(144, 24), rep(145, 24), rep(146, 24), rep(147, 24),
rep(148, 24), rep(149, 24), rep(150, 24),
rep(151, 24), rep(152, 24), rep(153, 24), rep(154, 24), rep(155, 24))

S = cbind(C, S)

fn = sprintf("%d_construct_samples_congruent.dat", sub)
write.table(S, fn, col.names=FALSE, row.names=FALSE)
```
unlink(cong_list_store[[1]])
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unlink(cong_list_store[[3]])
unlink(cong_list_store[[4]])
unlink(cong_list_store[[5]])
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unlink(cong_list_store[106])
unlink(cong_list_store[107])
unlink(cong_list_store[108])
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unlink(cong_list_store[110])

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unlink(cong_list_store[114])
unlink(cong_list_store[115])
unlink(cong_list_store[116])
unlink(cong_list_store[117])
unlink(cong_list_store[118])
For incongruent trials

# Run construct-samples

# For incongruent trials

for (sub in 1:1) {
    system(incong_list_run[[1]])
    system(incong_list_run[[2]])
    system(incong_list_run[[3]])
    system(incong_list_run[[4]])
    system(incong_list_run[[5]])
    system(incong_list_run[[6]])
    system(incong_list_run[[7]])
    system(incong_list_run[[8]])
    system(incong_list_run[[9]])
    system(incong_list_run[[10]])

    system(incong_list_run[[11]])
    system(incong_list_run[[12]])
    system(incong_list_run[[13]])
    system(incong_list_run[[14]])
    system(incong_list_run[[15]])
    system(incong_list_run[[16]])
    system(incong_list_run[[17]])
    system(incong_list_run[[18]])
    system(incong_list_run[[19]])
system(incong_list_run[[20]])

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system(incong_list_run[[46]])
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system(incong_list_run[[152]])
system(incong_list_run[[153]])
system(incong_list_run[[154]])
system(incong_list_run[[155]])

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S2 = read.table(incong_list_store[[2]], header = FALSE)
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S73 = read.table(incong_list_store[[73]], header = FALSE)
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S76 = read.table(incong_list_store[[76]], header = FALSE)
S77 = read.table(incong_list_store[[77]], header = FALSE)
S78 = read.table(incong_list_store[[78]], header = FALSE)
S79 = read.table(incong_list_store[[79]], header = FALSE)
S80 = read.table(incong_list_store[[80]], header = FALSE)
S81 = read.table(incong_list_store[[81]], header = FALSE)
S82 = read.table(incong_list_store[[82]], header = FALSE)
S83 = read.table(incong_list_store[[83]], header = FALSE)
S84 = read.table(incong_list_store[[84]], header = FALSE)
S85 = read.table(incong_list_store[[85]], header = FALSE)
S86 = read.table(incong_list_store[[86]], header = FALSE)
S87 = read.table(incong_list_store[[87]], header = FALSE)
S88 = read.table(incong_list_store[[88]], header = FALSE)
S89 = read.table(incong_list_store[[89]], header = FALSE)
S90 = read.table(incong_list_store[[90]], header = FALSE)
S91 = read.table(incong_list_store[[91]], header = FALSE)
S92 = read.table(incong_list_store[[92]], header = FALSE)
S93 = read.table(incong_list_store[[93]], header = FALSE)
S94 = read.table(incong_list_store[[94]], header = FALSE)
S95 = read.table(incong_list_store[[95]], header = FALSE)
S96 = read.table(incong_list_store[[96]], header = FALSE)
S97 = read.table(incong_list_store[[97]], header = FALSE)
S98 = read.table(incong_list_store[[98]], header = FALSE)
S99 = read.table(incong_list_store[[99]], header = FALSE)
S100 = read.table(incong_list_store[[100]], header = FALSE)
S101 = read.table(incong_list_store[[101]], header = FALSE)
S102 = read.table(incong_list_store[[102]], header = FALSE)
S103 = read.table(incong_list_store[[103]], header = FALSE)
S104 = read.table(incong_list_store[[104]], header = FALSE)
S105 = read.table(incong_list_store[[105]], header = FALSE)
S106 = read.table(incong_list_store[[106]], header = FALSE)
S107 = read.table(incong_list_store[[107]], header = FALSE)
S108 = read.table(incong_list_store[[108]], header = FALSE)
S109 = read.table(incong_list_store[[109]], header = FALSE)
S110 = read.table(incong_list_store[[110]], header = FALSE)

S111 = read.table(incong_list_store[[111]], header = FALSE)
S112 = read.table(incong_list_store[[112]], header = FALSE)
S113 = read.table(incong_list_store[[113]], header = FALSE)
S114 = read.table(incong_list_store[[114]], header = FALSE)
S115 = read.table(incong_list_store[[115]], header = FALSE)
S116 = read.table(incong_list_store[[116]], header = FALSE)
S117 = read.table(incong_list_store[[117]], header = FALSE)
S118 = read.table(incong_list_store[[118]], header = FALSE)
S119 = read.table(incong_list_store[[119]], header = FALSE)
S120 = read.table(incong_list_store[[120]], header = FALSE)

S121 = read.table(incong_list_store[[121]], header = FALSE)
S122 = read.table(incong_list_store[[122]], header = FALSE)
S123 = read.table(incong_list_store[[123]], header = FALSE)
S124 = read.table(incong_list_store[[124]], header = FALSE)
S125 = read.table(incong_list_store[[125]], header = FALSE)
S126 = read.table(incong_list_store[[126]], header = FALSE)
S127 = read.table(incong_list_store[[127]], header = FALSE)
S128 = read.table(incong_list_store[[128]], header = FALSE)
S129 = read.table(incong_list_store[[129]], header = FALSE)
S130 = read.table(incong_list_store[[130]], header = FALSE)

S131 = read.table(incong_list_store[[131]], header = FALSE)
S132 = read.table(incong_list_store[[132]], header = FALSE)
S133 = read.table(incong_list_store[[133]], header = FALSE)
S134 = read.table(incong_list_store[[134]], header = FALSE)
S135 = read.table(incong_list_store[[135]], header = FALSE)
S136 = read.table(incong_list_store[[136]], header = FALSE)
S137 = read.table(incong_list_store[[137]], header = FALSE)
S138 = read.table(incong_list_store[[138]], header = FALSE)
S139 = read.table(incong_list_store[[139]], header = FALSE)
S140 = read.table(incong_list_store[[140]], header = FALSE)

S141 = read.table(incong_list_store[[141]], header = FALSE)
S142 = read.table(incong_list_store[[142]], header = FALSE)
S143 = read.table(incong_list_store[[143]], header = FALSE)
S144 = read.table(incong_list_store[[144]], header = FALSE)
S145 = read.table(incong_list_store[[145]], header = FALSE)
S146 = read.table(incong_list_store[[146]], header = FALSE)
S147 = read.table(incong_list_store[[147]], header = FALSE)
S148 = read.table(incong_list_store[[148]], header = FALSE)
S149 = read.table(incong_list_store[[149]], header = FALSE)
S150 = read.table(incong_list_store[[150]], header = FALSE)

S151 = read.table(incong_list_store[[151]], header = FALSE)
S152 = read.table(incong_list_store[[152]], header = FALSE)
S153 = read.table(incong_list_store[[153]], header = FALSE)
S154 = read.table(incong_list_store[[154]], header = FALSE)
S155 = read.table(incong_list_store[[155]], header = FALSE)

S = rbind(S1, S2, S3, S4, S5, S6, S7, S8, S9, S10,
           S11, S12, S13, S14, S15, S16, S17, S18, S19, S20,
           S21, S22, S23, S24, S25, S26, S27, S28, S29, S30,
C = c(rep(1,16), rep(2,16), rep(3,16), rep(4,16), rep(5,16), rep(6,16), rep(7,16), rep(8,16), rep(9,16), rep(10,16),
rep(11,16), rep(12,16), rep(13,16), rep(14,16), rep(15,16), rep(16,16), rep(17,16),
rep(18,16), rep(19,16), rep(20,16),
rep(21,16), rep(22,16), rep(23,16), rep(24,16), rep(25,16), rep(26,16), rep(27,16),
rep(28,16), rep(29,16), rep(30,16),
rep(31,16), rep(32,16), rep(33,16), rep(34,16), rep(35,16), rep(36,16), rep(37,16),
rep(38,16), rep(39,16), rep(40,16),
rep(41,16), rep(42,16), rep(43,16), rep(44,16), rep(45,16), rep(46,16), rep(47,16),
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rep(51,16), rep(52,16), rep(53,16), rep(54,16), rep(55,16), rep(56,16), rep(57,16),
rep(58,16), rep(59,16), rep(60,16),
rep(61,16), rep(62,16), rep(63,16), rep(64,16), rep(65,16), rep(66,16), rep(67,16),
rep(68,16), rep(69,16), rep(70,16),
rep(71,16), rep(72,16), rep(73,16), rep(74,16), rep(75,16), rep(76,16), rep(77,16),
rep(78,16), rep(79,16), rep(80,16),
rep(81,16), rep(82,16), rep(83,16), rep(84,16), rep(85,16), rep(86,16), rep(87,16),
rep(88,16), rep(89,16), rep(90,16),
rep(91,16), rep(92,16), rep(93,16), rep(94,16), rep(95,16), rep(96,16), rep(97,16),
rep(98,16), rep(99,16), rep(100,16),
rep(101,16), rep(102,16), rep(103,16), rep(104,16), rep(105,16), rep(106,16), rep(107,16), rep(108,16), rep(109,16), rep(110,16),
rep(111,16), rep(112,16), rep(113,16), rep(114,16), rep(115,16), rep(116,16), rep(117,16), rep(118,16), rep(119,16), rep(120,16),
rep(121,16), rep(122,16), rep(123,16), rep(124,16), rep(125,16), rep(126,16), rep(127,16), rep(128,16), rep(129,16), rep(130,16),
rep(131,16), rep(132,16), rep(133,16), rep(134,16), rep(135,16), rep(136,16), rep(137,16), rep(138,16), rep(139,16), rep(140,16),
rep(141,16), rep(142,16), rep(143,16), rep(144,16), rep(145,16), rep(146,16), rep(147,16), rep(148,16), rep(149,16), rep(150,16),
rep(151,16), rep(152,16), rep(153,16), rep(154,16), rep(155,16))

S = cbind(C, S)

fn = sprintf("%d_construct_samples_incongruent.dat", sub)
write.table(S, fn, col.names=FALSE, row.names=FALSE)
}

unlink(incong_list_store[[1]])
unlink(incong_list_store[[2]])
unlink(incong_list_store[[3]])
unlink(incong_list_store[[4]])
unlink(incong_list_store[[5]])
unlink(incong_list_store[[6]])
unlink(incong_list_store[[7]])
unlink(incong_list_store[[8]])
unlink(incong_list_store[[9]])
unlink(incong_list_store[[10]])
unlink(incong_list_store[[11]])
unlink(incong_list_store[[12]])
unlink(incong_list_store[13])
unlink(incong_list_store[14])
unlink(incong_list_store[15])
unlink(incong_list_store[16])
unlink(incong_list_store[17])
unlink(incong_list_store[18])
unlink(incong_list_store[19])
unlink(incong_list_store[20])

unlink(incong_list_store[21])
unlink(incong_list_store[22])
unlink(incong_list_store[23])
unlink(incong_list_store[24])
unlink(incong_list_store[25])
unlink(incong_list_store[26])
unlink(incong_list_store[27])
unlink(incong_list_store[28])
unlink(incong_list_store[29])
unlink(incong_list_store[30])

unlink(incong_list_store[31])
unlink(incong_list_store[32])
unlink(incong_list_store[33])
unlink(incong_list_store[34])
unlink(incong_list_store[35])
unlink(incong_list_store[36])
unlink(incong_list_store[37])
unlink(incong_list_store[38])
unlink(incong_list_store[39])
unlink(incong_list_store[40])

unlink(incong_list_store[41])
unlink(incong_list_store[42])
unlink(incong_list_store[43])
unlink(incong_list_store[44])
unlink(incong_list_store[45])
unlink(incong_list_store[46])
unlink(incong_list_store[47])
unlink(incong_list_store[48])
unlink(incong_list_store[49])
unlink(incong_list_store[50])

unlink(incong_list_store[51])
unlink(incong_list_store[52])
unlink(incong_list_store[53])
unlink(incong_list_store[54])
unlink(incong_list_store[55])
unlink(incong_list_store[56])
unlink(incong_list_store[57])
unlink(incong_list_store[58])
unlink(incong_list_store[59])
unlink(incong_list_store[60])

unlink(incong_list_store[61])
unlink(incong_list_store[62])
unlink(incong_list_store[63])
unlink(incong_list_store[64])
unlink(incong_list_store[65])
unlink(incong_list_store[66])
unlink(incong_list_store[67])
unlink(incong_list_store[68])
unlink(incong_list_store[69])
unlink(incong_list_store[70])

unlink(incong_list_store[71])
Load EZ-DM estimates based on construct-samples data

# Load
EZ-DM estimates based on construct samples data for congruent trials
part2_EZ_construct_samples_cong <- read_excel("EZDM_construct_samples_congruent.xlsx")

EZ-DM estimates based on construct samples data for incongruent trials
part2_EZ_construct_samples_incong <- read_excel("EZDM_construct_samples_incongruent.xlsx")

# Select variables
From EZ-DM estimates based on construct samples data for congruent trials
part2_EZ_construct_samples_cong <- part2_EZ_construct_samples_cong[c("Participant",
    "driftRate",
    "boundSep",
    "nondecTime",
    "trialNum")]

From EZ-DM estimates based on construct samples data for incongruent trials
part2_EZ_construct_samples_incong <- part2_EZ_construct_samples_incong[c("Participant",
    "driftRate",
    "boundSep",
    "nondecTime",
    "trialNum")]

# Rename variables
For EZ-DM estimates based on construct samples data for congruent trials
setnames(part2_EZ_construct_samples_cong, old = c("driftRate",
    "boundSep",
    "nondecTime",
    "trialNum"),
    new = c("CONGdriftRate_CS",
    "CONGboundSep_CS",
    "CONGnondecTime_CS",
    "CONGtrialNum_CS"))

For EZ-DM estimates based on construct samples data for incongruent trials
setnames(part2_EZ_construct_samples_incong, old = c("driftRate", "boundSep", "nondecTime", "trialNum"),
new = c("INCONGdriftRate_CS", "INCONGboundSep_CS", "INCONGnondecTime_CS", "INCONGtrialNum_CS"))

# Remove rows containing negative NDT estimates as these denote poor model fit
# Exclude participants with negative NDT estimates arising from congruent trials
part2_EZ_construct_samples_cong <- part2_EZ_construct_samples_cong[part2_EZ_construct_samples_cong$CONGnondecTime_CS > 0, ]

# Exclude participants with negative NDT estimates arising from incongruent trials
part2_EZ_construct_samples_incong <- part2_EZ_construct_samples_incong[part2_EZ_construct_samples_incong$INCONGnondecTime_CS > 0, ]

# Merge into main data frame for Part 2 of the study
# Add EZ-DM estimates based on construct samples data for congruent trials
part2_data <- merge(part2_data, part2_EZ_construct_samples_cong, by.x = "newRID", by.y = "Participant")

# Then add EZ-DM estimates based on construct samples data for incongruent trials
part2_data <- merge(part2_data, part2_EZ_construct_samples_incong, by.x = "newRID", by.y = "Participant")

# Calculate combined (congruent and incongruent) DDM variables for EZ-DM estimates based on construct samples data
# Combined boundary separation
part2_data$COMBINEDboundSep_CS <- (as.numeric(part2_data$CONGboundSep_CS) + as.numeric(part2_data$INCONGboundSep_CS))/2

# Combined nondecision time
part2_data$COMBINEDnondecTime_CS <- (as.numeric(part2_data$CONGnondecTime_CS) + as.numeric(part2_data$INCONGnondecTime_CS))/2

# Combined drift rate
part2_data$COMBINEDdriftRate_CS <- (as.numeric(part2_data$CONGdriftRate_CS) + as.numeric(part2_data$INCONGdriftRate_CS))/2

Correlate empirical and construct-samples EZ-DM parameter estimates
# For congruent trials
round(cor(part2_data$CONGboundSep, part2_data$CONGboundSep_CS, use = "complete.obs"), 2)
## [1] 0.65
round(cor(part2_data$CONGdriftRate, part2_data$CONGdriftRate_CS, use = "complete.obs"), 2)
## [1] 0.69
round(cor(part2_data$CONGnondecTime, part2_data$CONGnondecTime_CS, use = "complete.obs"), 2)
## [1] 0.9

# For incongruent trials
round(cor(part2_data$INCONGboundSep, part2_data$INCONGboundSep_CS, use = "complete.obs"), 2)
## [1] 0.54
round(cor(part2_data$INCONGdriftRate, part2_data$INCONGdriftRate_CS, use = "complete.obs"), 2)
## [1] 0.74
round(cor(part2_data$INCONGnondecTime, part2_data$INCONGnondecTime_CS, use = "complete.obs"), 2)
## [1] 0.86

# For combined congruent and incongruent trials
round(cor(part2_data$COMBINEDboundSep, part2_data$COMBINEDboundSep_CS, use = "complete.obs"), 2)
## [1] 0.66
round(cor(part2_data$COMBINEDdriftRate, part2_data$COMBINEDdriftRate_CS, use = "complete.obs"), 2)
## [1] 0.63

Correlate empirical and construct-samples EZ-DM parameter estimates
# Plot correlations

## For congruent trials

CONGboundSep_plot <- ggplot(part2_data, aes(x=CONGboundSep, y=CONGboundSep_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Congruent boundary separation (r = 0.65)")

CONGdriftRate_plot <- ggplot(part2_data, aes(x=CONGdriftRate, y=CONGdriftRate_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Congruent drift rate (r = 0.69)")

CONGnondecTime_plot <- ggplot(part2_data, aes(x=CONGnondecTime, y=CONGnondecTime_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Congruent nondecision time (r = 0.9)")

## For incongruent trials

INCONGboundSep_plot <- ggplot(part2_data, aes(x=INCONGboundSep, y=INCONGboundSep_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Incongruent boundary separation (r = 0.54)")

INCONGdriftRate_plot <- ggplot(part2_data, aes(x=INCONGdriftRate, y=INCONGdriftRate_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Incongruent drift rate (r = 0.74)")

INCONGnondecTime_plot <- ggplot(part2_data, aes(x=INCONGnondecTime, y=INCONGnondecTime_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Incongruent nondecision time (r = 0.86)")

## For combined (congruent and incongruent) values

COMBINEDboundSep_plot <- ggplot(part2_data, aes(x=COMBINEDboundSep, y=COMBINEDboundSep_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Combined boundary separation (r = 0.66)")

COMBINEDdriftRate_plot <- ggplot(part2_data, aes(x=COMBINEDdriftRate, y=COMBINEDdriftRate_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Combined drift rate (r = 0.69)")

COMBINEDnondecTime_plot <- ggplot(part2_data, aes(x=COMBINEDnondecTime, y=COMBINEDnondecTime_CS)) +
  geom_point() +
  geom_smooth(method=lm, color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Combined nondecision time (r = 0.92)")

round(cor(part2_data$COMBINEDnondecTime, part2_data$COMBINEDnondecTime_CS, use = "complete.obs"), 2)
ggtitle("Combined drift rate \( (r = 0.78) \)"

COMBINEDnondecTime_plot <- ggplot(part2_data, aes(x=COMBINEDnondecTime, y=COMBINEDnondecTime_CS)) +
  geom_point() +
  geom_smooth(method=lm , color="red", se=TRUE) +
  labs(x = "Empirical", y = "Synthetic") +
  ggtitle("Combined nondecision time \( (r = 0.92) \)"

# Make big plot of all plots
ggarrange(CONGboundSep_plot, CONGdriftRate_plot, CONGnondecTime_plot,
           INCONGboundSep_plot, INCONGdriftRate_plot, INCONGnondecTime_plot,
           COMBINEDboundSep_plot, COMBINEDdriftRate_plot, COMBINEDnondecTime_plot,
           ncol = 3, nrow = 3)

## `geom_smooth()` using formula 'y ~ x'
## `geom_smooth()` using formula 'y ~ x'
## `geom_smooth()` using formula 'y ~ x'
## `geom_smooth()` using formula 'y ~ x'
## `geom_smooth()` using formula 'y ~ x'
## `geom_smooth()` using formula 'y ~ x'
## `geom_smooth()` using formula 'y ~ x'
## `geom_smooth()` using formula 'y ~ x'

\textit{Compare empirical and construct-samples mean values, etc. (absolute fit)}

# Manually enter values
# For congruent trials
## Empirical N
202
## construct-samples N
155
## Mean empirical proportion correct
97.17
## Mean construct-samples proportion correct
94.97
## Mean empirical RT
1.39
## Mean construct-samples RT
1.38
## Mean of SD empirical RT
0.29
## Mean of SD construct-samples RT
0.27
# For incongruent trials
## Empirical N
200
## construct-samples N
155
## Mean empirical proportion correct
92.59
## Mean construct-samples proportion correct
93.35
## Mean of SD empirical RT
0.27
## Mean of SD construct-samples RT
0.27

# Mean empirical RT
1.56

# Mean construct-samples RT
1.61

# Mean of SD empirical RT
0.31

# Mean of SD construct-samples RT
0.37

# t-tests
# Manually define t-test function to input mean and SD values rather than vectors

t.test2 <- function(m1,m2,s1,s2,n1,n2,m0=0,equal.variance=FALSE)
{
  if( equal.variance==FALSE )
  {
    se <- sqrt((s1^2/n1) + (s2^2/n2))
    # welch-satterthwaite df
    df <- ((s1^2/n1 + s2^2/n2)^2)/((s1^2/n1)^(2/(n1-1)) + (s2^2/n2)^2/(n2-1))
  }
  else
  {
    # pooled standard deviation, scaled by the sample sizes
    se <- sqrt((1/n1 + 1/n2) * ((n1-1)*s1^2 + (n2-1)*s2^2)/(n1+n2-2))
    df <- n1+n2-2
  }
  t <- (m1-m2-m0)/se
  dat <- c(m1-m2, se, t, 2*pt(-abs(t),df))
  names(dat) <- c("Difference of means", "Std Error", "t", "p-value")
  return(dat)
}

# Run t-test for congruent trials
round(t.test2(1.39, 1.38, 0.29, 0.27, 202, 155, m0=0, equal.variance = TRUE), 2)
## Difference of means           Std Error                   t             p-value
##                0.01                0.03                0.33                0.74

# Run t-test for incongruent trials
round(t.test2(1.56, 1.61, 0.31, 0.37, 200, 152, m0=0, equal.variance = TRUE), 2)
## Difference of means Std Error             t             p-value
##                -0.05                0.04            -1.38                0.17

# Remove all objects apart from the compiled data frame for part 2 of the study
rm(list=ls()[!ls() %in% c("part2_data")]) # Prevents my laptop from being overwhelmed

Inspect data

# Create a vector of dependent variables
part2_dependents <- c("COMBINEDboundSep", "COMBINEDnondecTime", "COMBINEDdriftRate")

# Create histograms for all dependent variables
ggplot(gather(subset(part2_data[c(part2_dependents)])), aes(value)) +
gem_histogram(bins = 15, fill = "#0c4c8a") +
facet_wrap(-key, scales = "free_x")
### Descriptive statistics

#### Work out gender ratio

```r
summary(part2_data$Gender)
```

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
<th>NA's</th>
</tr>
</thead>
<tbody>
<tr>
<td>count</td>
<td>102</td>
<td>47</td>
<td>1</td>
</tr>
</tbody>
</table>

#### Identify continuous variables to calculate descriptive statistics for

```r
```

#### Calculate descriptive statistics for all predictor variables

```r
summary(subset(part2_data[c(part2_vars)], text = T))
```

<table>
<thead>
<tr>
<th></th>
<th>IQ</th>
<th>Age</th>
<th>HypImp</th>
<th>InAtt</th>
<th>Tapping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Min.</td>
<td>76.0</td>
<td>4.33</td>
<td>0.0</td>
<td>0.0</td>
<td>-5.54</td>
</tr>
<tr>
<td>1st Qu.</td>
<td>91.0</td>
<td>5.92</td>
<td>6.2</td>
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<tr>
<td>Mean</td>
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<td>6.55</td>
<td>10.8</td>
<td>10.5</td>
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<tr>
<td>3rd Qu.</td>
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<td>7.42</td>
<td>16.0</td>
<td>15.8</td>
<td>0.00</td>
</tr>
<tr>
<td>Max.</td>
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<td>7.92</td>
<td>18.0</td>
<td>18.0</td>
<td>1.98</td>
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<th>Pursuit</th>
<th>Flanker</th>
<th>DCCS</th>
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<td>30.28</td>
<td>79.0</td>
<td>41.0</td>
</tr>
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</tr>
<tr>
<td>Median</td>
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<td>-0.48</td>
<td>100.0</td>
<td>97.5</td>
</tr>
<tr>
<td>Mean</td>
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<td>-1.45</td>
<td>99.1</td>
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<tr>
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<td>0.31</td>
<td>111.0</td>
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<tr>
<td>Max.</td>
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<td>1.46</td>
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<th>BDR</th>
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<th>COMBINEDdriftRate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Min.</td>
<td>71</td>
<td>79</td>
<td>64</td>
<td>8.154</td>
<td>0.0502</td>
</tr>
<tr>
<td>1st Qu.</td>
<td>101</td>
<td>79</td>
<td>64</td>
<td>8.209</td>
<td>0.1245</td>
</tr>
</tbody>
</table>
## Median :111  Median :104  Median :100  Median :0.225  Median :0.1386
## Mean   :111  Mean   :108  Mean   :101  Mean   :0.222  Mean   :0.1391
## 3rd Qu.:122  3rd Qu.:123  3rd Qu.:113  3rd Qu.:0.239  3rd Qu.:0.1530
## Max.   :148  Max.   :160  Max.   :150  Max.   :0.273  Max.   :0.2373
## NA's   :32  NA's :30  NA's :11

# COMBINEDnondecTime
## Min. :0.195
## 1st Qu.:0.518
## Median :0.699
## Mean :0.729
## 3rd Qu.:0.908
## Max. :1.617

# Calculate standard deviations
round(sapply(subset(part2_data[c(part2_vars)]), sd, na.rm = TRUE), 1)
##                   IQ            Age           HypImp          InAtt
##                12.0            0.9             6.0             5.9
##                   Tapping       Tracking       Pursuit       Flanker
##                 1.8             2.7             3.4             11.1
##                    DCCS           MRX          MRXproc            BDR
##                14.2           17.0           17.0            16.5
##  COMBINEDboundSep   COMBINEDdriftRate  COMBINEDnondecTime
##                0.0                0.0                0.3

# Calculate median absolute deviations
round(sapply(subset(part2_data[c(part2_vars)]), mad, na.rm = TRUE), 1)
##                   IQ            Age           HypImp          InAtt
##                13.3            1.1             5.9             5.9
##                   Tapping       Tracking       Pursuit       Flanker
##                 1.4             1.9             1.5             14.8
##                    DCCS           MRX          MRXproc            BDR
##                8.2            14.8            17.8            14.8
##  COMBINEDboundSep   COMBINEDdriftRate  COMBINEDnondecTime
##                0.0                0.0                0.3

Transformation
# Remove variables that aren't used in descriptive or inferential analysis
trans_data <- subset(part2_data, select = c("Age", "IQ", "HypImp",
                                          "Tapping", "Tracking", "Pursuit",
                                          "Flanker",
                                          "COMBINEDboundSep", "COMBINEDdriftRate", "COMBINEDnondecTime"))

# Transform remaining variables
trans_data <- huge.npn(trans_data)
# Conducting the nonparanormal (npn) transformation via shrunken ECDF....done.
trans_data <- as.data.frame(trans_data)
# Post transformation histograms
trans_data %>%
  gather() %>%
ggplot(aes(value)) +
  facet_wrap(~ key, scales = "free_x") +
  geom_histogram(bins = 10, fill = "#0c4c8a")
Correlational analyses

# Re-order variables

# Make correlation matrix
round(cor(trans_data, use = "pairwise.complete.obs", method = "pearson"), 2)

##                      Age    IQ Tapping Tracking Pursuit HypImp
## Age                 1.00  -0.41  -0.08  -0.03   0.12   0.19
## IQ                 -0.41  1.00   0.07   0.20   0.17  -0.04
## Tapping            -0.08  0.07  1.00   0.07   0.04  -0.11
## Tracking           -0.03  0.20  0.07  1.00   0.42  -0.02
## Pursuit            0.12  0.17  0.04  0.42  1.00   0.00
## HypImp             0.19  -0.04 -0.11  -0.02  0.00  1.00
## COMBINEDdriftRate  0.22  0.13  -0.05  0.15  0.16  -0.04
## COMBINEDboundSep   0.15  -0.06  -0.07  -0.11  0.07  -0.06
## COMBINEDnondecTime 0.45  0.17  0.10  -0.05  -0.16  -0.09
## Flanker            0.30  0.37  0.01  0.05   0.01  0.22

##                      COMBINEDdriftRate COMBINEDboundSep COMBINEDnondecTime
## Age               0.22   0.15   -0.45
## IQ                0.13  -0.06   0.17
## Tapping          -0.05  -0.07   0.10
## Tracking         -0.15  -0.11  -0.05
## Pursuit          -0.16   0.07  -0.16
## HypImp           -0.04  -0.06  -0.09
## COMBINEDdriftRate 1.00  -0.24  -0.12
## COMBINEDboundSep  0.24  1.00  -0.29
## COMBINEDnondecTime -0.12  -0.29  1.00
## Flanker           0.03  -0.01  0.12

## Flanker

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Confirmatory analyses

Non-decision time

# Perform multiple linear regression
part2_ndt_mlr <- lm(COMBINEDnondecTime ~ Tapping * HypImp,
                    data = trans_data, na.action = na.omit)

# Examine assumptions
check_model(part2_ndt_mlr)

# View results
summary(part2_ndt_mlr)

## Call:
# lm(formula = COMBINEDnondecTime ~ Tapping * HypImp, data = trans_data,
#    na.action = na.omit)
#
## Residuals:
## Min     1Q   Median     3Q    Max
## -2.1776 -0.6609 -0.0108  0.7214  2.5334
##
## Coefficients:
## Estimate Std. Error t value Pr(>|t|)
## (Intercept) -0.0157    0.0819  -0.19     0.85
## Tapping     0.1112    0.0833   1.33     0.18
## HypImp      -0.0938   0.0838  -1.12     0.26
## Tapping:HypImp -0.1632   0.1030  -1.58     0.12
##
## Residual standard error: 0.994 on 146 degrees of freedom
## Multiple R-squared:  0.0342, Adjusted R-squared:  0.0143
## F-statistic: 1.72 on 3 and 146 DF,  p-value: 0.165

**Boundary separation**

# Perform multiple linear regression
part2_boundary_mlr <- lm(COMBINEDboundSep ~ Tracking * HypImp, 
                         data=trans_data, na.action = na.omit)

# Examine assumptions
check_model(part2_boundary_mlr)

# View results
summary(part2_boundary_mlr)

## Call:
## lm(formula = COMBINEDboundSep ~ Tracking * HypImp, data = trans_data, 
##     na.action = na.omit)
##
## Residuals:
##     Min      1Q  Median      3Q     Max
##    -2.5034 -0.6896 -0.0206  0.7433  2.3847
##
## Coefficients:
##                  Estimate Std. Error t value Pr(>|t|)
## (Intercept) -0.000177   0.082006    0.00     1.00
## Tracking     0.110342   0.082714   1.33     0.18
## HypImp      -0.063266   0.084011  -0.75     0.45
## Tracking:HypImp -0.016574   0.091484  -0.18     0.86
##
## Residual standard error: 1 on 146 degrees of freedom
## Multiple R-squared:  0.0156, Adjusted R-squared:  -0.00465
## F-statistic: 0.77 on 3 and 146 DF,  p-value: 0.512

**Drift rate**

# Perform multiple linear regression
part2_driftrate_mlr <- lm(COMBINEDdriftRate ~ Tapping * HypImp + Tracking * HypImp, 
                           data=trans_data, na.action = na.omit)

# Examine assumptions
check_model(part2_driftrate_mlr)

# View results
summary(part2_driftrate_mlr)

## Call:
## lm(formula = COMBINEDdriftRate ~ Tapping * HypImp + Tracking * HypImp, 
##     data = trans_data, na.action = na.omit)
##
## Residuals:
##     Min      1Q  Median      3Q     Max
##    -2.5550 -0.743  0.049  0.701  2.407
##
## Coefficients:
##                  Estimate Std. Error t value Pr(>|t|)
## (Intercept) -0.00335    0.08241  -0.04     0.968
<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate 1</th>
<th>Estimate 2</th>
<th>Estimate 3</th>
<th>Estimate 4</th>
</tr>
</thead>
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<td>-0.75</td>
<td>0.457</td>
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<tr>
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<td>0.08433</td>
<td>-0.55</td>
<td>0.581</td>
</tr>
<tr>
<td>Tracking</td>
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<td>0.08268</td>
<td>1.88</td>
<td>0.063</td>
</tr>
<tr>
<td>Tapping:HypImp</td>
<td>-0.05793</td>
<td>0.10584</td>
<td>-0.55</td>
<td>0.585</td>
</tr>
<tr>
<td>HypImp:Tracking</td>
<td>0.08945</td>
<td>0.09309</td>
<td>0.96</td>
<td>0.338</td>
</tr>
</tbody>
</table>

---

Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error: 1 on 144 degrees of freedom
Multiple R-squared:  0.0352, Adjusted R-squared:  0.00175
F-statistic: 1.05 on 5 and 144 DF,  p-value: 0.39
Appendix M: ‘Journal of Child Psychology and Psychiatry’ Journal Submission Guidelines for Authors for Paper 2

Author Guidelines

Please read the Notes for Contributors guidance below for all types of contributions and styles of manuscript.

Why submit your article to The Journal of Child Psychology and Psychiatry?

- The leading, international journal covering both child and adolescent psychology and psychiatry;
- Provides an interdisciplinary perspective to the multidisciplinary field of child and adolescent mental health, though publication of high-quality empirical research, clinically-relevant studies and highly cited research reviews and practitioner review articles;
- Impact Factor 8.982 (2020): ISI Journal Citation Reports © (2020) 11/156 (Psychiatry) 8/143 (Psychiatry (Social Science)) 5/77 (Psychology) 1/78 (Psychology, Developmental);
- Ranked in the Top 20 journals in psychiatry and psychology by citation impact over the last decade (Thomson Reuters, Essential Science Indicators);
- Over 14,000 institutions with access to current content;
- Massive international readership; over one million articles downloaded every year (34% North America, 31% Europe, 10% Asia-Pacific);
- Quick turnaround times:
  - Decision on your paper in around 5 weeks (excluding reject without review decisions).
  - On average, articles are published online within 5 weeks of acceptace.
- Articles appear on Early View before the paper version is published – Click here; to see the Early View articles currently available online; Epub entries on PubMed and widely indexed/abstracted, including MEDLINE, EMBASE and ISI Citation Indexes;
- Every manuscript is assigned to 1 of the 19 decision editors specialising in a particular subject domain. Acceptance rate is around 16%;
- State of the art online submission site, simple and quick to use:- http://mc.manuscriptcentral.com/jcpp_journal; dedicated journal Editorial Office for easy, personal contact through the peer review and editorial process; proof tracking tool for authors.
- All papers published in JCPP are eligible for Panel A: Psychology, Psychiatry and Neuroscience in the Research Excellence Framework (REF);

Notes for Contributors

1. General
2. Authors’ professional and ethical responsibilities
   - Data Sharing

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Contributions from any discipline that further knowledge of the mental health and behaviour of children and adolescents are welcomed. Papers are published in English, but submissions are welcomed from any country. Contributions should be of a standard that merits presentation before an international readership.

Papers may assume either of the following forms:

- **Original articles**
  These should make an original contribution to empirical knowledge, to the theoretical understanding of the subject, or to the development of clinical research and practice. Adult data are not usually accepted for publication unless they bear directly on developmental issues in childhood and adolescence or the transition from adolescence to adulthood. Original articles should not exceed 6000 words, including title page, abstract, references, tables, and figures; the total word count should be given on the title page of the manuscript. Limit tables and figures to 5 or fewer double-spaced manuscript pages. It is possible to submit additional tables or figures as an Appendix for an online-only version. We strongly encourage you to keep the length of the manuscript within the word limit. If you would like to make an exceptional request to extend the length of your submission contact the editorial office (publications@acamh.org).

- **Review articles**
  Papers for this section can include systematic reviews, meta-analysis or theoretical formulations. There are three types of reviews: Annual Research Reviews, Research Reviews and Practitioners Reviews. These papers are usually commissioned. However, we also welcome proposals from authors which our specialist editors will review before inviting a submission. The papers should survey an important area of interest within a general field and, where appropriate, closely follow PRISMA guidelines. Practitioner Reviews and Research Reviews should normally be no more than 6000 words long (as original articles). Annual Research Reviews can be considerably longer with the length negotiated at the time of commission.

**Authors' professional and ethical responsibilities**
Submission of a paper to JCPP will be held to imply that it represents an original contribution not previously published (except in the form of an abstract or preliminary report); that it is not being considered for publication elsewhere; and that, if accepted by the Journal, it will not be published elsewhere in the same form, in any language, without the
consent of the Editors. When submitting a manuscript, authors should state in a covering letter whether they have currently in press, submitted or in preparation any other papers that are based on the same data set, and, if so, provide details for the Editors.

**Access to data and Data sharing**

If the study includes original data, at least one author must confirm that he or she had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

The journal encourages all authors to share the data and other artefacts supporting the results in the paper by archiving it in an appropriate public repository. Authors may provide a data availability statement, including a link to the repository they have used, in order that this statement can be published in their paper. Shared data should be cited.

More information is available [here](#).

All data must be made available on request of the editor-in-chief either before or after submission. Failure to do so before acceptance will result in rejection of the paper and after acceptance in retraction of the paper.

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**Authorship**

Authorship credit should be given only if substantial contribution has been made to the following:

- Conception and design, or collection, analysis and interpretation of data
- Drafting the article or revising it critically for important intellectual content, and final approval of the version to be published

The corresponding author must ensure that there is no one else who fulfils the criteria who is not included as an author. Each author is required to have participated sufficiently in the work to take public responsibility for the content.
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Recommended guidelines and standards
Randomised controlled trials
The Journal requires authors to conform to CONSORT 2010 (see CONSORT Statement) in relation to the reporting of randomised controlled clinical trials; also recommended is the Extensions of the CONSORT Statement with regard to cluster randomised controlled trials. In particular, authors of RCTs must include in their paper a flow chart illustrating the progress of subjects through the trial (CONSORT diagram) and the CONSORT checklist. The flow diagram should appear in the main paper, the checklist in the online Appendix. Trial registry name, registration identification number, and the URL for the registry should also be included at the end of the methods section of the Abstract and again in the Methods section of the main text, and in the online manuscript submission. The manuscript should include sample size calculation and should specify primary and secondary trial outcomes/endpoints.

Trials should be registered in one of the ICJME-recognised trial registries such as:

Australian New Zealand Clinical Trials Registry https://www.anzctr.org.au/
Clinical Trials http://www.clinicaltrials.gov
ISRCTN Register http://isrctn.org
Nederlands Trial Register http://www.trialregister.nl/trialreg/index.asp
UMIN Clinical Trials Registry http://www.umin.ac.jp/ctr

Trial registration must include a pre-registered, date stamped, publicly available protocol
setting out, at least, the research question, hypotheses, primary outcome and statistics plan. These requirements apply to all trials whatever their academic provenance (i.e., including trials of educational and social work interventions) or whether they include a clinical outcome (i.e., those trials that focus on a mechanism of action rather than symptoms or functional impairment retain the requirement for pre-registration). Authors must state whether the primary trial report is referenced and if they have identified the study as a secondary analysis of existing trial data.

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Systematic reviews should conform to the PRISMA guidelines. The journal strongly encourages the pre-registration of review protocols on publicly accessible platforms. From 2021 this will be mandatory.

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At this time the JCPP does not publish study protocols itself but actively encourages the practice to increase transparency and reproducibility of findings. This situation is under active review. Please click here for more details on our position.

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1. The manuscript should be double spaced throughout, including references and tables. Pages should be numbered consecutively. The preferred file formats are MS Word or WordPerfect, and should be PC compatible. If using other packages the file should be saved as Rich Text Format or Text only.

2. Papers should be concise and written in English in a readily understandable style. Care should be taken to avoid racist or sexist language, and statistical presentation should be clear and unambiguous. The Journal follows the style recommendations given in the Publication manual of the American Psychological Association (5th edn., 2001).

3. The Journal is not able to offer a translation service, but, authors for whom English is a second language may choose to have their manuscript professionally edited before submission to improve the English. A list of independent suppliers of editing services can be found here. All services are paid for and arranged by the author, and use of one of these services does not guarantee acceptance or preference for publication.
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The abstract should not exceed 300 words and should be structured in the following way with bold marked headings: Background; Methods; Results; Conclusions; Keywords; Abbreviations. The abbreviations will apply where authors are using acronyms for tests or abbreviations not in common usage.

Key points and relevance
All papers should include a text box at the end of the manuscript outlining the four or five key (bullet) points of the paper. These should briefly (80-120 words) outline what's known, what's new, and what's relevant.

Under the 'what's relevant' section we ask authors to describe the relevance of their work in one or more of the following domains - policy, clinical practice, educational practice, service development/delivery or recommendations for further science.

Headings
Articles and research reports should be set out in the conventional format: Methods, Results, Discussion and Conclusion. Descriptions of techniques and methods should only be given in detail when they are unfamiliar. There should be no more than three (clearly marked) levels of subheadings used in the text.

Acknowledgements
These should appear at the end of the main text, before the References.

Correspondence to
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References
The JCPP follows the text referencing style and reference list style detailed in the Publication manual of the American Psychological Association (5th edn.).

References in text
References in running text should be quoted as follows: Smith and Brown (1990), or (Smith, 1990), or (Smith, 1980, 1981a, b), or (Smith & Brown, 1982), or (Brown & Green, 1983; Smith, 1982).

For up to five authors, all surnames should be cited in the first instance, with subsequent occurrences cited as et al., e.g. Smith et al. (1981) or (Smith et al., 1981). For six or more authors, cite only the surname of the first author followed by et al. However, all authors should be listed in the Reference List. Join the names in a multiple author citation in running text by the word ‘and’. In parenthetical material, in tables, and in the References List, join the names by an ampersand (&). References to unpublished material should be avoided.
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Full references should be given at the end of the article in alphabetical order, and not in footnotes. Double spacing must be used.

References to journals should include the authors’ surnames and initials, the year of publication, the full title of the paper, the full name of the journal, the volume number, and inclusive page numbers. Titles of journals must not be abbreviated and should be italicised.

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2. Include only those items that are relevant and ensure that all appendices, figures, tables etc included are referenced in the manuscript in chronological order.
3. Label and cite the items presented in the Supporting Information as – Appendix S1, Figure S1, and Table S1 etc in the order of their appearance.
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