2011

A meta-analysis of response inhibition and Stroop interference control deficits in adults with traumatic brain injury (TBI)

Aneta Dimoska-Di Marco

Skye McDonald
University of New South Wales, s.mcdonald@unsw.edu.au

Michelle Kelly
University of New South Wales, mkelly@psy.unsw.edu.au

Robyn Tate
University of Sydney, rtate@med.usyd.edu.au

Stuart Johnstone
University of Wollongong, sjohnsto@uow.edu.au

Publication Details

Dimoska-Di Marco, A., McDonald, S., Kelly, M., Tate, R. & Johnstone, S. (2011). A meta-analysis of response inhibition and Stroop interference control deficits in adults with traumatic brain injury (TBI). Journal of Clinical and Experimental Neuropsychology, 33 (4), 471-485.

A meta-analysis of response inhibition and Stroop interference control deficits in adults with traumatic brain injury (TBI)

Publication Details
Dimoska-Di Marco, A., McDonald, S., Kelly, M., Tate, R. & Johnstone, S. (2011). A meta-analysis of response inhibition and Stroop interference control deficits in adults with traumatic brain injury (TBI). Journal of Clinical and Experimental Neuropsychology, 33 (4), 471-485.

A meta-analysis of performance in inhibitory control paradigms in adults with traumatic brain injury (TBI)

Aneta Dimoska-Di Marco[1], Skye McDonald[1], Michelle Kelly[1], Robyn Tate[2], Stuart Johnstone[3]

[1] School of Psychology, University of NSW

[2] Rehabilitation Studies Unit, Northern Clinical School, Faculty of Medicine, University of Sydney

[3] School of Psychology, University of Wollongong

Journal of Clinical and Experimental Neuropsychology ,

33 (4), 471-485

Abstract

Clinical features of traumatic brain injury (TBI) such as impulsivity suggest an impairment in inhibitory control processes, and a recent surge in studies suggest this is likely. This meta-analysis consolidates findings to-date in adults following TBI across mild to severely-injured groups, focusing on 'effortful' inhibition processes: response inhibition and response interference control. The review studies using well-defined paradigms of response inhibition (N = 20) such as the Go/Nogo task, Sustained-Attention-To-Response Tasks, Stop-signal tasks and Conners' CPT, and the Stroop Colour-Word Task (N = 19 studies) as a measure of response interference control. A small-to-moderately sized average effect was observed for reduced inhibitory control across 41 effect sizes in 989 participants with TBI, compared with 969 controls. However, the effect was larger in studies measuring response inhibition performance, supporting a deficit in this particular process following TBI. Stroop interference control was reduced in the TBI compared to control group largely when studies used the outcome measure 'total time on task', but not 'RT per trial' or 'number of stimuli'. This latter finding suggests factors other than interference control, such as fatigue and arousal, may underlie poor performance on such tasks, and it highlights the importance of adopting a cautious approach when selecting among various Stroop task versions and measures to distinguish TBI deficits.

(abstract words = 212)

A meta-analysis of performance in inhibitory control paradigms in adults with traumatic brain injury (TBI)

Executive dysfunction is a well-established outcome of traumatic brain injury (TBI) causing an inability to adapt and regulate behaviour to changing environmental demands (Stuss, Hugenholtz, Richard, LaRochelle, Poirier & Bell 1985; Levin & Kraus 1994; Tate 1999). Such behavioural change following TBI has been explained by models involving two dissociable systems: loss of inhibitory control and loss of drive (Tate 1999). Inhibitory control, in particular, is an important function of the frontal-subcortical executive system allowing us to suppress, interrupt or delay an activated behaviour or cognitive course of action (Starkstein 1997; Aron, Robbins & Poldrack 2004). Clinical features of TBI suggest a failure in this mechanism with frequent reports of an inability to inhibit impulsive and habitual behaviour, and socially inappropriate responses such as inappropriate touching and verbal disinhibition (Rao & Lyketsos 2000). Brain imaging findings during tasks involving cognitive control also support an inhibition deficit (Soeda, Nakashima, Okumura, Kuwata, Shinoda & Iwama 2005) with reduced activation in prefrontal regions in patients with mild (McAllister, Saykin, Flashman, Sparling, Johnson, Guerin, Mamourian, Waeaver & Yanofsky 1999; McAllister, Sparling, Flashman, Guerin, Mamourian & Saykin 2001) and moderate-to-severe TBI (Perlstein, Cole, Demery, Seignourel, Dixit, Larson & Briggs 2004).

TBI involves a myriad of diffuse and focal brain damage caused by the *coup/contra-coup* (acceleration/deceleration) force on the brain following an external blunt force to the head. Focal contusional damage is common across the poles and inferior surface of the frontal lobes, both the dorsolateral and orbitofrontal areas, the inferior and lateral surfaces of the temporal lobes, as well as cortical regions around Sylvian fissures and limbic areas (Bigler 2001; Bonne, Gilboa, Louzoun, Kempf-Sherf, Katz, Fishman, Ben-Nahum, Krausz, Bocher, Lester, Chisin & Lerer 2003). Indirect functional impairment can also occur due to

lesions that are external to the frontal lobes but located in regions with a high number of afferent or efferent connections to frontal regions, general cortical atrophy and diffuse axonal injury (Adams, Graham & Jennett 2001; Bigler 2001; Khan, Baguley & Cameron 2003). This diffuse pattern of brain damage means localised lesions to frontal regions are not a necessary precursor to frontal executive dysfunction in this group (Rieger & Gauggel 2002).

While poor inhibitory control following TBI is but one of many cognitive sequalae, it nevertheless presents as an important and promising function to focus on as it has been shown to be amenable to functional changes arising from learning and rapid plasticity of neural networks (Chambers, Bellgrove, Stokes, Henderson, Garavan, Robertson & Mattingley 2006; Kelly, Hester, Foxe, Shpaner & Garavan 2006; Chambers, Garavan & Bellgrove). From a clinical perspective, this means that identifying deficient inhibition processes may highlight areas for the development of targeted remediation programs. The utility of this line of research has been identified by researchers, with a recent surge in studies examining inhibitory control deficits in TBI since 2000 (e.g. Felmingham, Baguley & Green 2004; Perlstein, Larson, Dotson & Kelly 2006; Larson, Kaufman, Schmalfuss & Perlstein 2007). However, there has been no attempt to consolidate inhibition findings to-date. Past meta-analyses studies have broadly examined frontal executive functioning in adults with moderate to severe TBI, reporting a range of effect sizes from small 0.2 – 0.3 (Belanger, Curtiss, Demery, Lebowitz & Vanderploeg 2005; Frenchmen, Fox & Mayberry 2005) to large 0.9 (Mathias & Wheaton 2007). In a meta-analysis of mild TBI patients, while moderate-to-large effect sizes were obtained across all cognitive domains, the largest effect observed was for cognitive flexibility/abstraction (d = .72) (Zakzanis, Leach & Kaplan 1999).

Such studies include a vast array of measures of executive function such as the Trail Making Test, Wisconsin Card Sorting Test and Tower of London. Although these tasks have been purported to access inhibitory control, they also evoke multiple processes beyond

inhibition, and therefore, have poor construct validity for measuring inhibition (Halperin, McKay, Matier & Sharma 1994; Milich, Hartung, Martin & Haigler 1994). Experimental paradigms, on the other hand, are designed to isolate inhibition and provide a more direct measurement of inhibitory control. Commonly used paradigms include the Go/Nogo task (e.g. Roche, Dockree, Garavan, Foxe, Robertson, O'Mara, Roche, Dockree, Garavan, Foxe, Robertson & O'Mara 2004), Stop-signal task (Logan 1994), the Sustained Attention to Response Task (SART) (Robertson, Manly, Andrade, Baddeley & Yiend 1997), and some versions of the Continuous Performance Task (CPT) (Duncan, Kosmidis & Mirsky 2005). Each of these tasks measures an overt, effortful, expression of inhibition involving the suppression of an activated motor response (Nigg 2000). *Response inhibition*, typically measured as the number of inhibition failures (i.e. failure to stop a response when required), has been found to be impaired in adults with TBI in a number of studies, compared with controls (e.g. Roche et al. 2004; O'Keeffe, Dockree, Moloney, Carton & Robertson 2007). Other measures of response inhibition include the speed of the inhibition process, termed the Stop-signal Reaction Time (SSRT)(Logan 1994).

A cognitive form of inhibition known as interference control is commonly measured by the Stroop Colour-Word task (e.g. Stuss, Floden, Alexander, Levine & Katz 2001). This task typically includes 'control' sub-tasks involving Colour-Naming and/or Word-Reading that measure attention and processing speed, and a third sub-task involving colour-naming when the colour word and ink are incongruent. Effortful inhibition at covert, cognitive level is required in the third sub-task to suppress the competing automatic response in favour of the correct response (Nigg 2000). There have, however, been inconsistent findings with some finding Stroop interference control does not differentiate TBI and controls (Stuss et al. 1985; Ponsford & Kinsella 1992; Batchelor, Harvey & Bryant 1995) and others showing significant differences (Larson, Kaufman, Schmalfuss, Perlstein, Larson, Kaufman, Schmalfuss &

Perlstein 2007; Schroeter, Ettrich, Schwier, Scheid, Guthke, von Cramon, Schroeter, Ettrich, Schwier, Scheid, Guthke & von Cramon 2007). Mathias et al. (2007) performed a meta-analysis of performance on the Stroop task in adults with severe TBI, among other tasks involving attention, and found an overall small-to-moderately sized effect (d = 0.36) across two studies examining speed in the Colour-Word incongruent subtask (Marsh & Knight 1991; Spikeman, Deelman & van Zomeren 2000) but larger effect sizes across four studies when scores for speed and inhibition failures were combined (d = 0.87) (Ponsford & Kinsella 1992; Bate, Mathias & Crawford 2001; Bate, Mathias & Crawford 2001; Felmingham, Baguley, Green, Felmingham, Baguley & Green 2004; Rios, Perianez & Munoz-Cespedes 2004). However, in this analysis, effects were based on stand-alone measures of performance in the incongruent subtask, and not as a difference score relative to a control condition. As performance on incongruent Stroop trials reflects a number of perceptual and cognitive processes, as well as processing speed, it is vital that difference interference scores are calculated in reference to a control 'reading' condition in order to isolate processes related to inhibitory control (Lansbergen, Kenemans & van Engeland 2007).

Evidence that response inhibition and interference control load on the same latent process suggest they rely on a global inhibitory mechanism (Verbruggen, Liefooghe, Notebaert & Vandierendonck 2005). Further support for this position comes from findings of common areas of neural activation including the right dorsolateral prefrontal and anterior cingulated cortices, the right inferior frontal and superior medial frontal regions (Cabeza, Grady, Nyberg, McIntosh, Tulving, Kapur, Jennings, Houle & Craik 1997; Bush, Frazier, Rauch, Seidman, Whalen, Jenike, Rosen & Biederman 1999; Aron & Poldrack 2005) (Rubia, Russell, Overmeyer, Brammer, Bullmore, Sharma, Simmons, Williams, Giampietro, Andrew & Taylor 2001; Wager, Sylvester, Lacey, Nee, Franklin & Jonides 2005; Nee, Wager & Jonides 2007). However, unique neural contributions have also been observed with

activation of the thalamus and right parietal cortex during response inhibition and the left inferior frontal gyrus during interference control (Wager et al. 2005). Furthermore, studies have shown different developmental patterns for response inhibition and interference control (Bedard, Nichols, Barbosa, Schachar, Logan & Tannock 2002; van den Wildenberg & van der Molen 2004; Johnstone, Pleffer, Barry, Clarke & Smith 2005), and dissociable patterns of impairment in clinical disorders such as ADHD (Johnstone, Barry, Markovska, Dimoska & Clarke 2008). This review may provide further insight into this issue.

It has also been suggested that response processing speed problems may account for poor performance on the inhibitory component of the Stroop task (Ponsford & Kinsella 1992; Mathias & Wheaton 2007). Slow response processing is a well-established effect in adults with TBI (Ponsford & Kinsella 1992; Frenchmen et al. 2005; for a review see Mathias & Wheaton 2007) and may be due to diffuse axonal injury leading to reduced interconnections between networks and a disruption of effective neural transmission (Felmingham et al. 2004). Indeed, performance on each of the 'control' sub-tests of the Stroop task, reflecting attention and response processing speed rather than inhibition, has been shown to discriminate mild TBI from healthy participants better than many other neuropsychological tests (Bohen, Jolles & Twijnstra 1992; Bate, Mathias & Crawford 2001; for a review see Mathias & Wheaton 2007).

As apparent from this review, there is significant variability in the outcomes between inhibition studies. Characteristics of TBI injury, including severity and time since injury, as well as participant characteristics of age and gender may account for some of this variability. A meta-analysis examining cognitive performance across mild to severe TBI patients and across different neuropsychological domains found significant differences in effect sizes dependent on time-since-injury (Schretlen & Shapiro 2003). In contrast, other meta-analytic reviews have found no such relationship for executive functions following severe TBI

(Mathias & Wheaton 2007) (Frenchmen et al. 2005). Mild TBI has been shown to be associated with smaller-sizes on neuropsychological measures (d = .24) when compared with moderate-to-severe TBI (d = .74) (Schretlen & Shapiro 2003), and recovery of mild cognitive impairment following mild TBI is common within the first three months (Ponsford 2000; Frenchmen et al. 2005). Furthermore, gender differences in response inhibition have been found with females showing greater control than males (Ray-Li, Huang, Constable & Sinha 2006). Finally, it is well known that cognitive functioning declines with increasing age (Park 2000), in some ways mimicking the effects of TBI. Therefore, these variables will require consideration as possible moderators of inhibition effect sizes.

The aim of this meta-analysis is to provide a consolidation of findings from studies to-date examining deficits in inhibitory control following TBI across mild to severely-injured groups, focusing on processes *response inhibition* based upon several tasks and *response interference control* as measured specifically by the Stroop interference effect. The latter focus will provide an update to an existing meta-analysis (Mathias & Wheaton 2007), and report effect sizes as relative measures in reference to a control condition. A further aim is to determine whether TBI impairs speed of response processes in the inhibition paradigms reviewed, and whether this may account for inhibition impairments.

Method

Search strategy and inclusion criteria

A systematic review of the literature was performed to identify studies that have examined the effects of traumatic brain injury on inhibitory control. A staged process was used to locate relevant articles using PsycINFO and MEDLINE (including Premedline) from

Jan 1980 to Dec 2008 using the search terms and their variants 'traumatic brain injury' or 'closed head injury', and 'brain injury', 'head injury', and 'concussion', combined with 'response inhibition', 'interference', and a search for inhibitory paradigms using commonly known terms (i.e. 'nogo', 'continuous performance', 'sustained attention to response', 'stop-signal', 'stroop'). Searches were performed independently by two researchers (AD and MK) and then results were combined. Only articles in English were examined. Abstracts and unpublished studies were excluded.

To be included in the analysis, studies had to meet several criteria. First, participants had to be adults (aged over 18 years) with a traumatic brain injury as a result of an external blow to the head, and there had to be some report in the study evidencing brain injury including loss of or altered consciousness, presence of confusion or post-traumatic amnesia (PTA), amnesia or objective neurological findings (Tate, McDonald & Lulham 1998). Head injuries which only caused momentary loss of consciousness without any following altered consciousness, drowsiness or dazing were not included. Additionally, adults with lesions to the brain due to cerebrovascular disorders, tumors, or other neurological conditions were also excluded from this study. Furthermore, studies that only included children aged less than 18 years in their sample were not included in the current analysis as they may present with different cognitive sequalae due to developmental influences (Borg, Holm, casidy, Peloso, carroll, von Holst, Paniak & Yates 2004). Second, participants with TBI had to be compared to a non-brain-injured control group (no single-case studies) using a parametric design, which is a necessary precursor for estimating effect sizes (*d*) (Hunter & Schmidt 1990). Third, studies had to report sufficient statistical information in order to allow for calculation of effect sizes (see *Data extraction and statistical analysis*). Fourth, TBI and control participants had to have performed an 'inhibition task' and have been compared on an 'inhibition measure', as outlined in the next section.

Inhibition paradigms

While a number of different paradigms have been used by researchers purporting to measure inhibitory control, many of these have also been criticised for their poor construct validity as they evoke a number of other processes beyond inhibition (Halperin et al. 1994; Milich et al. 1994). Furthermore, there have been a number of taxonomic issues with the definition of inhibition. Therefore, we adopted Nigg's (2000) definition of effortful inhibition and restricted our meta-analysis to commonly-used paradigms measuring (a) response inhibition (e.g. the Go/Nogo task, stop-signal task) and (b) the Stroop-Colour Word task as a measure of response interference control, although it is acknowledged that there are a host of paradigms that may evoke these and other processes of inhibition.

A 'response inhibition' task was defined as one where a frequent (prepotent) motor response is inhibited intermittently on infrequent trials (i.e. on <50% of trials) (de Zubicaray, Andrew, Zelaya, Williams & Dumanoir 2000; Ramautar, Kok & Ridderinkhof 2004; Dimoska & Johnstone 2008). These tasks included the Go/Nogo task (e.g. Roche et al. 2004), the Sustained Attention to Response task (SART) (Robertson et al. 1997), and the Conners' Continuous Performance Task (CPT) (Conners 1995), which involve serial single-trial presentations of infrequent Nogo stimuli interspersed throughout Go stimuli. Other versions of the CPT, such as Gordan's CPT (McAllister, Flashman, Harker Rhodes, Tyler, Moore, Saykin, McDonald, Tosteson & Tsongalis 2008), were not included as they require an infrequent response, failing to establish a prepotent response tendency. The stop-signal task was also included as it has been shown to be the most overt measure of response inhibition, and involves the infrequent inhibition of an already executed response to a choice-RT task (Logan 1994).

The Stroop Colour-Word task is a well-established paradigm of 'response interference control' in that a competing automatic response set must be inhibited in order to execute the effortful correct response (e.g. Stuss et al. 2001). Tasks had to include a 'control' sub-task of either colour-naming, word-reading or both, and the inhibitory colour-word subtest, in order to isolate inhibition. The difference in performance between this and the control tasks is known as the 'interference effect' (Bate et al. 2001; Mathias & Wheaton 2007), with larger difference scores reflecting greater interference (i.e. poorer inhibitory control). In some cases, colour words are printed in the same coloured ink and, conversely, a facilitatory 'congruency effect' is observed with a reduction in reaction time.

Outcome Measures

Inhibition outcome measures included the rate of Nogo errors (i.e. failures to stop the motor response), Stop-signal Reaction Time (SSRT; i.e., the latency of the inhibition process) and the Stroop interference score. Performance in the Stroop Colour-Word task was measured in three different forms, including (a) total time on task (secs), (b) number of stimuli completed within 100 secs, or (c) mean reaction time within trials (secs).

Eight out of 19 Stroop-task studies reported an interference score, however, as studies differed in their approach to calculating this score, we calculated an interference score for all studies to ensure consistency by subtracting the score (either number correct or reaction time) for control trials (i.e. 'colour naming', or 'word naming' trials, and in absence of either 'congruent colour naming trials') from the score for incongruent trials for each group (van Mourik, Oosterlaan & Sergeant 2005). The standard deviation of the interference score was calculated with the formulae: $SD_{INT} = \sqrt{(2 \text{ x } \{[SD_C^2 + SD_I^2]/2\} \text{ x } (1 - r)}$, where SD_C and SD_I are the pooled standard deviation across the control and TBI groups for congruent (or neutral)

trials and incongruent trials, respectively, and *r* is the Pearson coefficient of correlation between performance on these two trial-types (Lansbergen et al. 2007). There is some conjecture as to what *r* should be set. We adopted the value of *r* = .954, derived from a past study of adult participants (Kenemans, Wieleman, Zeegers & Verbaten 1999) and used by Lansbergen et al. (2007). Estimating interference control using group-level scores when individual scores are not available is a common technique employed in meta-analyses (van Mourik et al. 2005; Lansbergen et al. 2007). A larger interference score means greater interference from conflicting response sets, or poorer inhibitory control. Likewise, a larger rate of Nogo errors and SSRT indicate poorer inhibitory control.

Response processing outcome measures included mean reaction time (MRT) to Go stimuli in the response inhibition tasks (secs) and to Go stimuli on trials where there was no stop-signal in the stop-signal task (secs). In the Stroop Colour-Word task, response processing was measured for (a) neutral colour-naming stimuli, (b) neutral word-reading stimuli, and (c) congruent coloured word-reading (in either 'total time on task', 'number of stimuli completed', or MRT).

Data extraction and statistical analysis

The following information was obtained for all studies and served as the basis of the meta-analysis: (i) number of participants in each group, (ii) group means, (ii) within-group standard-deviations, and (iii) between-group *t/F* and *p* statistics. For three studies, means and standard deviations were estimated from figures (Felmingham et al. 2004; Seignourel, Robins, Larson, Demery, Cole & Perlstein 2005; Schroeter et al. 2007). Furthermore, some minor transformations to reported data were necessary in order to calculate effect sizes: (i) where standard errors were reported, these were converted to standard deviations, (ii)

descriptive variables (e.g. time since injury, PTA, period of unconsciousness, number of patients per severity classification) were converted to the same scale of measurement. Data were extracted by one researcher (AD) and then verified by a second researcher (MK), both of whom were experienced with inhibitory control research.

Effect sizes (*d*) were calculated separately for the inhibition and response processing measures (Hunter & Schmidt 1990). The effect size *d* was calculated as the TBI group mean minus the control group mean divided by the pooled standard deviation (Hunter & Schmidt 1990; Zakzanis 2001), representing the standardised difference between the two groups within each study. However, effect sizes (*d*) have been shown to be upwardly biased when based on a small sample size, therefore, a bias correction was performed using Hedge's formulae (Hedges & Olkin 1985, see p. 81), and were then weighted by the inverse variance according to the formulae $1/v_d = ([n_T + n_C]/n_T n_C) + (d^2/2[n_T + n_C - 2])$ (Hedges & Olkin 1985). If means and standard deviations were not provided for a between-group analysis, we converted *t* and *F* statistics to *d* using the formulae $d = 2t/(\sqrt{df})$ or $(2\sqrt{F})/(\sqrt{df_{error}})$ (Zakzanis 2001). A positive effect size indicated a greater inhibition deficit or slower response processing in the TBI group. The size of the effect was defined using Cohen's (1988) thresholds (i.e. small 0.1-0.3, moderate 0.30 – 0.50, large > 0.5). For the overall inhibition analysis, where participants completed different versions of the same task (e.g. fixed and random SART) or different conditions within a task (e.g. O'Keeffe, Dockree & Robertson 2004; O'Keeffe et al. 2007), individual effect sizes were calculated and then averaged together. In contrast, where multiple severity groups within TBI were presented, effect sizes were calculated separately for each group and treated as separate trials. Additionally, for a more detailed analysis of the Stroop interference effect, individual effect sizes were also calculated if different versions of the task were used within a study.

We identified potential outliers in effect sizes using funnel plots and replaced one outlier from the interference control studies (d = 7.23) with the mean (Hunter & Schmidt 1990). Average weighted 'Inhibition' effect sizes were calculated across all inhibition measures and then separately for the Nogo error rate and for the Stroop interference effect. Average 'Response Processing' effect sizes were calculated across all Go MRT measures in the response inhibition tasks and for either of the 'control' sub-tasks (a) neutral colour-naming stimuli, or (b) neutral word-reading stimuli in the Stroop Colour-Word task, and then separately for the response inhibition tasks, the Stroop 'control' subtasks and the Stroop 'congruent' subtask.

Comprehensive Meta Analysis (Version 2.2.048, www.Meta-Analysis.com) was used to estimate and compare average effect sizes derived through both fixed effects and random effects models. Effects sizes derived using the fixed effects model were initially examined, with homogeneity analyses performed by calculating an overall *Q*-statistic to test whether effect sizes differed significantly between studies. The *Q*-statistic has a chi-square distribution with k – 1 degrees of freedom, where k is the number of effect sizes. Where *Q* for overall inhibition and response processing constructs was significant, moderator variables (see below) were analysed to determine whether they contributed to effect size variability by examining differences between defined sub-groups of moderator variables using the fixed-effects ANOVA-analog of the *Q*-statistic (i.e. $Q_{between}$ and Q_{within}). When $Q_{between}$ is significant it suggests systematic differences between studies due to the moderator variable, and when Q_{within} is significant it suggests within-subject heterogeneity due to an additional random component. In these cases, effect sizes from the random effects model were reported for each sub-group within the moderator variable (Lansbergen et al., 2007). Statistical analyses were also performed to determine whether effect sizes significantly differed from zero using two-tailed *Z*-tests. A *Fail-safe N* analysis, which predicts the number of

unpublished 'lost' studies that would be required to render the effect size of this meta-analysis insignificant, was also calculated using a weighted approach (Rosenberg 2005).

Moderator Variables

Given that response inhibition and interference control are considered separate but related processes of inhibitory control (Nigg 2000), we anticipated that there may be differences in inhibition effect sizes between studies using response inhibition paradigms and studies using the Stroop task. Therefore, *inhibition type* was examined as a moderator variable. Further moderator variables included, *Age:* The mean of the TBI and control groups was averaged together and then split into two sub-groups using a median-split (median age = 30.7 years). All studies reported age. *Severity of TBI injury:* Thirty-seven studies reported the breakdown in severity of the TBI sample (this was based on either the GCS or PTA). Where the sample consisted either wholly or predominantly (estimated as > 65%) of patients classified with mild TBI, the study was coded as 'mild' (1). Where the sample consisted of an equal proportion of patients classified as mild or moderate, the study was coded as 'moderate' (2). Finally where the sample consisted of predominantly moderate to severe patients or severe and greater, the study was coded as 'severe' (3). There were no studies where the whole sample was classified as moderately severe. A further eight studies reported a mean GCS or range which allowed the severity of the overall group to be determined, and this was coded accordingly. *Time since injury:* The mean of time elapsed since injury to testing was reported in 31 studies, which was converted to months to ensure consistency across studies, and two sub-groups were formed using a median-split (median = 38.6 months).

In the detailed analysis of Stroop-task studies only, *outcome measure* was examined as a moderator variable. Each study/task was coded for the type of dependent variable used to measure the interference effect and included 'total time' to read a set number of stimuli, 'RT per trial', and 'total number' of stimuli read within a set time. Other moderator variables were not examined for separate inhibition sub-types.

Results

The literature search initially yielded 173 potential studies. Of these, 92 studies were rejected from initial analysis of abstracts for not measuring inhibitory control, and another 42 studies were excluded after a detailed analysis, with these reasons outlined in Table 1.

_____

Table 1 about here

_____

Therefore, there were 39 studies that satisfied the inclusion criteria for adults with TBI (see asterisked studies in the reference list), and included 20 response inhibition tasks: five Go/Nogo tasks, 11 Sustained-Attention-To-Response Tasks, three Stop-signal tasks, one Conners' CPT, and a further 19 studies using Stroop Colour-Word tasks measuring response interference control. The earliest studies were published in 1989 with a recent surge of 21 studies in the last 5 years since 2003.

The characteristics of the identified studies and their participants are outlined in Table 2. A total of 989 adults with TBI (58% males; Mean Age: 31.2 years) and 969 non-brain-injured control adults (54% males; Mean Age: 31.5 years) contributed to the meta-analysis.

All but four studies reported that control participants were matched to participants in the TBI group, with the majority matching by age, gender and education. Twenty-seven out of 41 studies reported a mean or range for the Glasgow Coma Score (GCS)(Mean: 8.3, Range: 3 to 15) and 27 reported the duration of post-traumatic amnesia (PTA)(Mean: 31.7 days, Range: < 1 hour to 210 days). Average time since injury was 65.9 months (SD: 146.3 months; Range: 0.1 – 833.3 months). Fifteen out of 41 studies reported the number of patients presenting with focal frontal lesions. Of the five studies that included only mildly impaired patients, three studies reported that patients had been tested within the first month (Potter, Jory, Bassett, Barrett & Mychalkiw 2002; Chan & Chan 2005; DeHaan, Halterman, Langan, Drew, Osternig, Chou, van Donkelaar, DeHaan, Halterman, Langan, Drew, Osternig, Chou & van Donkelaar 2007), one study tested their sample between 12-34 months following injury (Bohen et al., 1995), and the fifth did not provide this information (Stewart & Tannock 1999).

_____

Table 2 about here

_____

Overall Effect of Inhibitory Control

Table 3 provides a summary of the effect sizes for inhibition and response processing in the response inhibition paradigms and Stroop tasks. Table 4 reports the inhibition and response processing effect sizes derived in both the fixed and random effects models, though fixed model effects sizes are initially reported below.

_____

Tables 3 and 4 about here

_____

It is clear that the fixed effects model provides the more conservative estimates. The overall weighted mean effect (*d*) of TBI on inhibitory processes was 0.30 (95% confidence interval 0.20 – 0.39) based on 41 effect sizes and differed significantly from zero ($Z = 6.0$, $p<.001$). A *Fail-safe N* analysis revealed that 517 unpublished 'lost' studies would be required to render the effect size insignificant.

As the homogeneity analysis was significant ($Q(40) = 464.2$, $p<.001$), an analysis of moderators was performed and revealed that time since injury ($Q_{between}$ (1) < 1), severity of injury ($Q_{between}$ (2) = 3.3, $p = 0.192$) and age ($Q_{between}$ (1) < 1) did not explain the variability. In contrast, inhibition type (i.e. response inhibition or Stroop interference) significantly accounted for the variability in effect sizes ($Q_{between}$ (1) = 21.7, $p < .001$), although within-group variance was also significant (Q_{within} (39) = 442.5, $p < .001$).

Inhibition Effect Sizes by Inhibition Type

Response inhibition. Individual effect sizes in the 20 response inhibition studies ranged from 0 to 1.32, yielding a weighted mean effect size of 0.50 (95% confidence interval 0.37 – 0.63) that differed from zero ($Z = 7.6$, $p<.001$). The homogeneity analysis was non-significant, indicating similar effect sizes across studies, $Q(19) = 29.2$, $p >.05$. There were no studies showing a negative effect size.

We also separately examined the rate of Nogo errors in six Stroop task studies that reported this measure, revealing an average weighted effect size of 0.62 (95% confidence

interval 0.34 – 0.89) that was significantly greater than zero (Z = 4.5, p<.001), Q(5) = 9.1, p >.05.

Stroop interference effect. Although there were 19 Stroop task studies, there were 21 effect sizes due to two studies separately examining two different TBI patient groups (Felmingham et al. 2004; Seignourel et al. 2005). Individual effect sizes ranged from -3.72 to 4.60, yielding a small average weighted effect size of 0.05 (95% confidence interval -0.09 – 0.19) that did not significantly differ from zero (Z < 1). Eight effect sizes were negative, indicating effects in the opposite direction to expectations (i.e. TBI groups showed better performance than controls).

Due to a large number of studies employing more than one version of the Stroop task and the large variability observed in effect sizes between studies, we calculated individual effect sizes for each sub-task within a study. The weighted mean effect size was again small at 0.05 and non-significant (Z<1) across 27 effect sizes (95% confidence interval -0.08 – 0.17) and the homogeneity analysis was significant at Q(26) = 547.7, p <.001. A closer examination revealed that the outcome measure differentiated between studies, $Q_{between}$(2) = 266.1, p <.001. However, the pooled within-groups variance was also significant, Q_{within}(24) = 281.7, p <.001. Thus, variability in Stroop interference effect sizes was due to both systematic differences in the outcome measure employed by studies, as well as an additional random component. Applying the effect sizes from the random effects model revealed a significant effect size for 'total time on task' (d = 1.4, 95% CI 0.8 – 1.9, Z = 4.7, p<.001) but not 'RT per trial' (d = -0.8, 95% CI -1.7 – 0.1, Z = -1.8, p=.08) or 'total number of stimuli' (d = -0.9, 95% CI -1.9 – 0.1, Z = -1.7, p=.08).

Overall Effect of Response Processing

The overall weighted mean effect (*d*) of TBI on response processes was 0.58 based on 31 effect sizes (95% confidence interval 0.48 to 0.68) and was significantly greater than zero ($Z = 11.1$, $p<.001$). A *Fail-safe N* analysis revealed that 241 unpublished 'lost' studies would be required to render the effect size insignificant. A correlational analysis between inhibition and Go MRT effect sizes revealed no relationship across the whole sample ($r = -0.295$, $p>.05$), suggesting inhibitory performance was independent of response processing.

The homogeneity analysis was also significant, $Q(30) = 123.8$, $p <.001$. A moderator analysis revealed that variability in effect sizes was not explained by time since injury ($Q_{between}(1) < 1$) or age ($Q_{between}(1) = 2.8$, $p =.095$). However, severity of injury significantly accounted for the variance between studies ($Q_{between}(2) = 10.0$, $p <.01$), although within-subject heterogeneity was also significant ($Q_{within}(24) = 111.8$, $p <.001$). Therefore, applying the random-model effect sizes revealed significant effect sizes for moderate (d = 0.45, 95% CI 0.22 – 0.68, Z = 3.8, p<.01) and, to a greater degree, severely injured patients (d = 0.84, 95% CI 0.50 – 1.2, Z = 4.8, p<.001) but not mildly injured patients (d = 0.20, 95% CI -0.27 – 0.68, Z < 1). Furthermore, there were differences observed between inhibition types ($Q_{between}(1) = 37.4$, $p <.001$) and within-subject heterogeneity was also significant ($Q_{within}(29) = 86.4$, $p <.001$).

Response Processing by Inhibition Type

We also examined response processing in the two types of inhibition tasks.

Response inhibition. Effect sizes for Go MRT ranged from -0.39 to 1.30 and had a mean weighted effect size of 0.31 (95% confidence interval 0.18 to 0.44) that was significantly

greater than zero (Z = 4.5, p<.001), Q(16) = 36.9, p <.05. Three effect sizes were negative, and there was no relationship between response and inhibition effect sizes (r = -0.01, p>.10).

Stroop 'control' sub-tasks. Effect sizes ranged from 0.1 to 2.4 and had a weighted mean effect size of 0.96 was observed (95% confidence interval 0.80 to 1.12) that differed from zero (Z = 11.8, p<.001), Q(13) = 49.5, p <.001. There were no negative effect sizes.

Examining separate Stroop tasks, the outcome measure differentiated between studies $Q_{between}(2) = 33.5$ p <.001. However, the pooled within-groups variance was also significant, $Q_{within}(14) = 56.0$, p <.001. Applying the random effects model effect sizes revealed significant effect sizes for 'total time' (d = 0.92, 95% CI 0.52 – 1.34, Z = 4.5, p<.001), 'RT per trial' (d = 2.87, 95% CI 1.43 – 4.30, Z = 3.9, p<.001), and for 'total number' (d = 1.28, 95% CI 0.97 – 1.59, Z = 8.1, p<.001).

Stroop 'congruency effect'. A weighted mean effect size of 0.78 was observed across 13 studies, 95% confidence interval 0.61 to 0.96 and significantly differed from zero (Z = 9.7, p<.001), Q(12) = 47.9, p<.001. There were too few studies per outcome measure category to perform any moderator analyses.

Discussion

The findings in this meta-analysis revealed a small-to-moderate effect for an overall deficit in inhibitory control across 41 effect sizes in 989 participants with TBI ranging from mild to very severe, compared with 969 controls. Response processing across tasks was associated with an average moderate-to-large effect size. The majority of studies matched TBI and control groups for age, gender, and education suggesting these variables were unlikely to account for differences between groups.

Examining inhibition of a prepotent response in the response inhibition paradigms separately revealed a moderately-sized deficit in adults with TBI, as measured by the inhibition rate or SSRT. Automatic and habitual responding following TBI is common, and indeed these patients show little or no difficulty with automatic or well-learned tasks (Loken et al., 1995; Levin et al., 1988). Problems became evident when effortful processing is demanded to stop a course of action that is made inappropriate by changing circumstances. Inefficient response inhibition can arise when inhibition fails to activate or if it is slow to activate, or if the response process is relatively too fast and/or variable (Logan 1994). As TBI adults showed overall slower response speed than controls, and the measure was unrelated to the inhibition effect, it is unlikely that response speed contributed to impaired inhibition. Consequently, this analysis suggests that the difficulty lies with inhibition itself, failing to activate or activating too slowly. A further aim of this study was to update an existing meta-analysis of Stoop Colour-Word task performance (Mathias & Wheaton 2007) by providing a more specific examination of the Stroop interference effect, a sub-process of inhibitory control. The small and non-significant effect we found for Stroop interference remained even when separate effect sizes were estimated for different versions of the task (i.e. total time, total number, RT/trial). This finding suggests that the ability to control interference from competing response tendencies may not necessarily be impaired in adults with TBI. Heterogeneity in effect sizes between studies was observed and appeared to reflect, in part, methodological differences. Indeed, a closer examination of the type of dependent variable employed revealed significant differences with larger effect sizes for studies that employed 'total time' taken to the complete the Stroop task, while effect sizes were negative for 'RT per trial' and 'number of stimuli completed'.

The implications of this finding are particularly noteworthy for researchers selecting the type of Stroop task and/or outcome measure when conducting research. If requiring a

measure that best discriminates patients with TBI from controls, then 'total time' on task appears to be useful. However, 'total time' taken as a measure of performance across a task is unlikely to isolate interference control per se, rather being influenced by other factors such as response speed, fatigue and arousal (Lansbergen et al. 2007). 'RT per trial' is likely to be a more direct and less variable measure of interference control. However, this measure demands use of a computer-administered task and this is not routine practice in clinical settings due to variability in commercially-available programs and a lack of normative data. Nevertheless, these findings do highlight the lack of discriminability, and perhaps utility, in versions of the Stroop task that require counting the total number of items completed within a 45 second period. Indeed, Lansbergen et al (2007) obtained a similar finding in a meta-analysis of the Stroop interference effect in children with ADHD, however, here both 'time' outcomes (including both 'total time' and 'RT per trial') presented with a larger effect size (d = 1.1) than the 'number of items' outcome (d = -0.007). A direct examination of effect sizes dissociated by the 'type' was not feasible as there were too few studies for each of the many variations.

It should also be noted that the large differences in effect sizes between that observed for response inhibition (i.e., the complete cessation of a motor response) and Stroop interference control indicate that the two processes of inhibition reflected in these measures (Nigg, 2000) may not necessarily rely on a global inhibitory mechanism (Verbruggen et al. 2005). This finding supports studies showing different developmental patterns for these two inhibition processes (Bedard et al. 2002; van den Wildenberg & van der Molen 2004; Johnstone et al. 2005) and dissociable patterns of impairment in clinical disorders such as AD/HD (Johnstone et al. 2008).

Effect sizes for response processing (Go MRT) were moderate-to-large in the inhibition paradigms. The inhibition effect (d = 0.50) and response speed effect (d = 0.31) in

the response inhibition paradigms may be considered equally discriminating. In contrast, the effect sizes for the control sub-tasks of the Stroop paradigm (i.e. Word-Reading and Colour-Naming) provided a much better discriminating effect size (d = 0.96) than the interference effect (d = 0.05). This finding is in line with a past meta-analysis performed by Mathias and Wheaton (2007) and indicates that speed-of-processing is a greater problem for adults with TBI performing the Stroop paradigm than is interference control. Speed of processing deficits have consistently been shown to underlie cognitive dysfunction particularly following severe TBI (Bate et al., 2001; Felmingham et al., 2004; Mathias & Wheaton, 2007). Indeed, we found that injury severity was a significant predictor of heterogeneity in effect sizes between studies for response processing, in line with a past meta-analytic review (Schretlen & Shapiro 2003). Patients with severe injury showed a greater impairment than those with a moderate injury, while patients with mild TBI did not show an effect size that differed from zero.

A positive relationship was observed between moderator variable 'injury severity' and impairment in response processing. While this is in contrast to Mathias et al.'s review (2007), the finding is in line with the notion that more severe injury may be associated with greater DAI, which impairs speed of processing (Felmingham et al. 2004). In contrast, no relationship was observed between 'injury severity' and overall inhibition, in line with meta-analytic reviews examining general executive dysfunction (Frenchmen et al. 2005) and individual experimental studies showing no relationship between inhibitory performance and severity of injury (Loken, Thornton, Otto & Long 1995). Similarly, we found no relationships between 'time since injury' and inhibition or response processing comparing groups using a median split of 38.6 months, suggesting no recovery in observed impairments. Belanger (2005) found significant differences between adults with mild TBI measured acutely (i.e. <90 days), who showed significant impairment across 7 out of 8 cognitive

domains measured (including executive functioning), with almost full recovery in those measured postacutely (i.e. ≥90 days). One reason for this difference is that there was a skew in our review towards studies examining moderate-to-severe patients and few mild cases by comparison, therefore, we caution that these findings should be examined further

It should be noted that the focus of this review is narrow in addressing only one cognitive sequalae out of a number of the multiple cognitive deficits suffered by TBI. However, this focus is justified when one considers the degree and persistence of disinhibitory behaviours suffered by adults with TBI, causing significant social implications (McDonald 2005). Furthermore, our narrow search strategy may have excluded studies that have employed inhibitory tasks but that have described them in different terms (e.g. attention). Two tasks that have been debatably linked with interference control in some studies but that were excluded here include the negative priming and task-switching paradigms. Interference control in these tasks likely occurs at a perceptual-discrimination level of stimulus processing, inhibiting attention towards distractors rather than the inhibition of an inappropriate response (Nigg 2000). This restricted approach was necessary considering the variability and ambiguity in definitions of response inhibition, ensuring our findings specifically pertained to effortful forms of response inhibition as defined within Nigg's (2000) framework.

Conclusions

Importantly, this meta-analytic review found that the ability to inhibit an inappropriate response is a moderately-sized problem following TBI, and this was unrelated to speed of response processing, supporting a deficit in the response inhibition process following TBI. In contrast, there was a great deal of variation in the extent to which Stroop interference control

was affected by TBI injuries, though this finding was likely due to the different task designs and outcome measures employed between studies. For example, the effect was only noteworthy when studies used the outcome measure ‘total time on task’, which reflects other factors contributing to deteriorated performance including arousal and fatigue. Our findings highlight the need for researchers to adopt a cautious approach when choosing measures of response interference control among various Stroop task versions when distinguishing TBI deficits.

Table 1. Number of excluded papers by grounds for exclusion

Ground	Response Inhibition	Interference Control
Insufficient data	6	4
Children only	5	0
Not a prescribed response inhibition or interference control task	7	1
No control group	2	5
Same group as another study - no new measures	1	2
Non-parametric design	2	2
No inhibition measures reported	0	2
Population group sustained repetitive injuries	0	1
No control trial type for interference control measure	NA	6

Note: Multiple grounds may apply to one study.

Table 2. Participant characteristics of included studies.

Study		TBI			Controls			TBI Injury Severity				
		N	Mean Age	No. Males	*N*	Mean Age	No. Males	Mean GCS (SD and/or Range [R]) (months unless otherwise stated)	Mean PTA (SD and/or Range [R]) (days unless otherwise stated)	Code	Mean time since injury in months (SD and/or Range [R])	No. with frontal lesions
1	Braun et al. (1989)	22	31.04	NR	22	30.59	NR	NR	21.8 (5-42)	3	80.6 (R 10-204)	NR
2	Draper et al. (2008)	51	41.98	28	43	42.3	24	7.4 (SD 4.3, R 3-15)	26.3 (SD 24.7, R 0.1 to 99)	3	127 (R 120-144)	34
3	O'Keefe et al. (2007)	18	33.3	17	18	32.9	16	R 3-14	NR	3	39 (SD 26.97, R 9-97)	9
4	O'Keefe et al. (2004)	16	32.1	12	16	29.8	11	NR	NR	3	66 (SD 62)	NR
5	Dockree et al. (2006)	29	28.66	26	32	29.69	24	NR	NR	3	34.81 (SD 21.94)	17
6	Dockree et al. (2004)	10	35.5	8	10	38.7	8	NR	NR	3	78 (SD 79)	NR
7	Gagnon et al. (2006)	30	36.3	20	30	36.3	20	8.2 (R 3-13)	Retrograde amnesia 3.48 mnths (SD 9.0, R 0-36)	2	40 (SD 38)	20
8	Whyte et al. (2006)	26	36	23	35	37	30	<12	NR	3	Median 34 (3.6-410)	NR
9	McAvinue et al. (2005)	18	28	15	16	26.75	13	NR	NR	3	36.7 (SD 38.8, R 2-132)	NR
10	Chan et al. (2005)	51	42.9	42	51	41.7	43	14 (R 7-15)	0 (0-24hr)	1	25 (R 2-127)	10
11	Roche et al. (2004)	7	35.9	5	8	40	5	NR	NR	.	NR	NR
12	Armillo (2003)	14	35	6	14	35	6	R 3-15	R 1-37 days	2	60 (SD 12, R 30-66)	NR
13	Manly et al. (2003)	19	28.74	16	16	26.75	13	NR	NR	3	NR	NR
14	Hales (1999)	24	18	15	24	18.7	12	NR	x-30 days (all >2 hrs)	.	2-12 years	NR
15	Robertson et al. (1997)	22	34.2	16	17	39.8	11	11.1 (SD 4.1)	NR	3	9-18	NR
16	Rugg et al. (1989)	20	26.7	18	20	26.8	20	<9	>48 hrs	3	NR	NR

17	De Haan et al. (2007)	17	21.8	9	17	22.6	8	Disorientation > 15 mins (Grade 2)	NR	1	2 days	NR
18	Reiger & Gauggell (2002)	26	40.8	21	26	42.8	21	NR	NR	2	Median 8	16
19	Stewart & Tannock (1999)	42	33.9	26	42	33.9	26	NR	NR	1	NR	NR
20	Incoccia 2004	18	32.3	15	36	32.6	25	<8	NR	3	39 (SD 38)	NR
21	Cantin et al. (2007)	10	37	8	10	38.4	8	7.6 (SD 2.6, R 5-13)	15.9 (SD 6.6, R 7-25)	3	5.4 (SD 8.4, R 1-28.7)	8
22	Larson et al. (2007)	19	30.4	15	21	25.4	12	4.5 (SD 1.8, R 3-8)	35.3 (SD 28.4, R 7-120)	3	11.3 (SD 7.5, R 2-29)	11
23	Schroeter et al. (2007)	10	26.2	9	10	NR (matched to TBI group)	9	6.7 (SD 4.6, R 3-14)	63.4 (SD 65.9, R 6-199)	3	38.6 (SD 25.2, R 7-96)	0
24	Seignourel et al. (2005) Mild Group	20	34	10	24	35.9	12	14 (R 14-14)	1.2 hrs (SD 0.01)	2	62.9 (SD 11.4, R 1-137)	NR
25	Seignourel et al. (2005) Mod Group	26	40.7	17	24	35.9	12	5 (SD 0.9, R 3-7)	664.6 hrs (175.0)	3	110 (SD 22.6, R 1.5-144)	NR
26	Perlstein et al. (2006)	11	45	4	11	41.4	4	GCS score <9	40.1 (SD 49.3, R 2.5-180)	3	99 (SD 111, R 21-384)	NR
27	Summers et al. (2006)	10	38.6	7	10	38.6	4	4.6 (SD 2.3, R 3-8)	72.0 (SD 57.5, R 14-210)	3	52.90 (SD 29.05, R 24-112)	NR
28	Felmingham et al. (2004) Mixed Group	10	29	8	10	32	7	9.5 (SD 2.6)	19.3 (SD 11)	3	32.9 (SD 12.6)	7
29	Felmingham et al. (2004) Diffuse Group	10	25	6	10	32	7	5 (SD 1.8)	35.7 (SD 20.2)	3	59.9 (SD 34.8)	4
30	Rios et al. (2004)	29	28.9		30	25.85	NR	4.9 (SD 1.3)	46 (SD 31.9)	3	12.07 (SD 7.37)	NR
31	Potter et al. (2002)	24	32	16	24	31.4	14	R 13-15	(0 secs - 30 mins)	1	6 (R 3-36)	0
32	Bate et al. (2001)	35	28.9	28	35	30.2	20	5.7 (SD 3.1)	43.2 (SD 37.9)	3	833.3 (SD 990.3)	24 out of 33
33	Simpson & Schmitter-Edgecombe (2000)	18	32.28		18	32.18	NR	NR	NR	NR	NR	NR
34	Spikeman et al. (1996)	60	28.8	38	60	28.5	36	NR	9.5 days (SD: 10.4, R 1-51)	3	32.7 (SD: 9.9, R 15-55)	NR

35	Ponsford et al. (1992)	47	23.4	29	30	25.4	24	4.7 (SD 2.1, R: 3-9)	39.6 (SD: 34.8, R: 7-168)	3	3.7 (SD: 2.5, R: 0.9-11.8)	17 out of 42
36	Bohnen et al. (1992)	44	24.5	NR	44	23.4	NR	15	10 (R 6-14)	2	NR	NR
37	Batchelor et al. (1995)	35	25.6	NR	35	25.6	NR	R 13-15	<1 hr to 48 hrs	2	6.4 days (R 2-11)	NR
38	Bohnen et al. (1995)	11	27.2	6	11	28.1	6	15	<60 mins	1	12-34 mnths (Med 21)	NR
39	Killam et al. (2005)	5	22.6	3	8	21.4	5	Concussion Index Score 4.7 (SD 3.6)	NR	.	2.5 (SD 6)	NR
40	Vakil et al. (1995)	25	27.04	21	27	23.37	NR	7 (R 3-15)	NR	3	5.2 (R 0.8-14)	NR
41	Hales (1999)	24	18	15	24	18.7	12	NR	x-30 days (all >2 hrs)	.	2-12 years	NR

Note: SD = Standard Deviation, R = Range, NR = not reported, '.' = could not be determined. Injury Severity Code: Participants considered 1 = Mostly Mild, 2 = Mild to Moderate, 3 = Moderate to Severe. Notes: Studies 1 – 20 measure response inhibition, 20 – 41 measure Stroop interference control.

Table 3. A summary of effect sizes and variance for the inhibition and response processing constructs in response inhibition paradigms (1 - 20) and Stroop tasks (21 - 41).

			Inhibition		Response	
Trial	Study	Task and Inhibition Rate (%)	Effect size (d)	Variance	Effect size (d)	Variance
1	Braun et al. (1989)	Go/Nogo 50%	0.73	1.00	1.30	0.11
2	Draper et al. (2008)	SART 11%	0.48	0.50	0.11	0.04
3	O'Keefe et al. (2007)	SART 11%	1.13	0.33	NM	NM
4	O'Keefe et al. (2004)	SART 11%	1.21	0.25	NM	NM
5	Dockree et al. (2006)	SART 11%	0.63	0.20	-0.39	0.07
6	Dockree et al. (2004)	SART 11%	1.32	0.17	NM	NM
7	Gagnon et al. (2006)	Go/Nogo 20%	0.47	0.14	0.72	0.07
8	Whyte et al. (2006)	SART 11%	0.00	0.13	0.81	0.07
9	McAvinue et al. (2005)	SART 11%	1.01	0.11	0.44	0.12
10	Chan et al. (2005)	SART 11%	0.25	0.10	0.73	0.04
11	Roche et al. (2004)	Go/Nogo 6%	0.98	0.09	0.34	0.27
12	Armillo (2003)	SART (NR)	0.72	0.08	0.29	0.14
13	Manly et al. (2003)	SART 11%	0.95	0.08	0.46	0.12
14	Hales (1999)	Conners CPT 10%	0.01	0.07	0.50	0.09
15	Robertson et al. (1997)	SART 11%	0.84	0.07	-0.29	0.11
16	Rugg et al. (1989)	Go/Nogo 50%	0.77	0.06	0.19	0.10
17	De Haan et al. (2007)	Countermanding Saccade Stop-signal Task 33%	0.04	0.06	-0.33	0.12
18	Reiger & Gauggell (2002)	Visual/Auditory Stop-signal 25%	0.14	0.06	0.10	0.08
19	Stewart & Tannock (1999)	Visual/Auditory Stop-signal 25%	0.49	0.05	0.05	0.05
20	Incoccia (2004)	Go/Nogo 50%	0.22	0.05	0.24	0.08
21	Cantin et al. (2007)	Stroop Card list	0.91	0.05	2.43	0.35
22	Larson et al. (2007)	Stroop PC single trial	0.73	0.05		
23	Schroeter et al. (2007)	Stroop PC single trial	0.42	0.04	0.56	0.21
24	Seignourel et al. (2005) *Mild Group*	Stroop Card list	-0.64	0.04	NM	NM
25	Seignourel et al. (2005) *Mod Group*	Stroop Card list	-0.69	0.04	NM	NM
26	Perlstein et al. (2006)	Stroop Cued PC single trial	-3.72	0.04	2.86	0.37
27	Summers et al. (2006)	Stroop Card list	0.56	0.04	0.79	0.22
28	Felmingham et al. (2004) *Mixed Group*	Stroop Card list	2.97	0.04	NM	NM
29	Felmingham et al. (2004) *Diffuse Group*	Stroop Card list	4.60	0.03	NM	NM
30	Rios et al. (2004)	Stroop Card list	-2.37	0.03	1.72	0.09
31	Potter et al. (2002)	Stroop PC single trial	-0.51	0.03	NM	NM
32	Bate et al. (2001)	Stroop Card list	-1.49	0.03	1.23	0.07
33	Simpson & Schmitter-Edgecombe (2000)	Stroop PC single trial	-0.02	0.03	NM	NM
34	Spikeman et al. (1996)	Stroop Card list	0.26	0.03	0.78	0.04
35	Ponsford et al. (1992)	Stroop Card list	-3.03	0.03	1.18	0.06
36	Bohnen et al. (1992)	Stroop Card list	0.76	0.03	0.36	0.05
37	Batchelor et al. (1995)	Stroop Card list	1.26	0.03	0.67	0.06
38	Bohnen et al. (1995)	Stroop Card list	1.47	0.03	0.20	0.18
39	Killam et al. (2005)	Stroop Card list	-1.00	0.03	0.07	0.33

40	Vakil et al. (1995)	Stroop Card list	3.88	0.03	1.95	0.11
41	Hales (1999)	Stroop PC single trial	2.06	0.02	0.86	0.09

Note: NM = not measured; NR = not reported; SART = Sustained-attention-to-response task; CPT = continuous performance task; PC = personal computer.

Table 4. Meta-analysis statistics for inhibitory control and response processing effects using fixed-effects and random-effects models.

		Fixed-effects				Random-effects				Heterogeneity Test		
	K	*Mean*	95% CI of mean	*Z*	*p*	*Mean*	95% CI of mean	*Z*	*p*	*Q*	*df*	*p*
Inhibitory Control												
Overall	41	0.3	0.2 - 0.39	6.1	0.000	0.42	0.09 - 0.75	6.1	0.000	464.2	40	0.000
Response Inhibition	20	0.5	0.37 - 0.63	7.6	0.000	0.54	0.37 - 0.70	6.4	0	29.2	19	0.063
Interference Control	21	0.05	-0.09 - 0.19	0.7	0.508	0.27	-0.4 - 0.93	0.8	0.432	413.3	20	0.000
Response Process												
Overall	31	0.26	0.15 - 0.36	4.9	0.000	0.32	0.03 - 0.61	2.2	0.031	222.3	30	0.000
Response Inhibition	17	0.31	0.18 - 0.45	4.5	0.000	0.31	0.10 - 0.52	2.9	0.004	36.9	16	0.002
Interference Control	14	0.18	0.02 -0.34	2.2	0.027	0.37	-0.26 - 0.99	1.2	0.249	183.9	13	0.000

References

Adams, J. H., Graham, D. I. & Jennett, B. (2001). The structural basis of moderate disbaility after traumatic brain damage. *Journal of Neurology, Neurosurgery & Psychiatry* **71**: 521-524.

*Armilio, M. L. (2003). Electrophysiological correlates of response inhibition and error processing: The effects of strategic manipulation, feedback and traumatic brain injury, Armilio, Maria Luisa: U Toronto, Canada.

Aron, A. R. & Poldrack, R. A. (2005). The cognitive neuroscience of response inhibition: Relevance for genetic research in attention-deficit/hyperactivity disorder. *Biological Psychiatry* **57**(11): 1285-1292.

Aron, A. R., Robbins, T. W. & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences* **8**(4): 170-177.

*Batchelor, J., Harvey, A. G. & Bryant, R. A. (1995). Stroop colour-word test as a measure of attentional deficit following mild head injury. *Clinical Neuropsychologist* **9**(2): 180-186.

Bate, A. J., Mathias, J. L. & Crawford, J. R. (2001). The covert orienting of visual attention following severe traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology* **23**: 386-398.

*Bate, A. J., Mathias, J. L. & Crawford, J. R. (2001). Performance on the test of everyday attention and standard tests of attention following severe traumatic brain injury. *The Clinical Neuropsychologist* **15**: 405-422.

Bedard, A.-C., Nichols, S., Barbosa, J. A., Schachar, R., Logan, G. D. & Tannock, R. (2002). The development of selective inhibitory control across the life span. *Developmental Neuropsychology* **21**(1): 93 - 111.

Belanger, H. G., Curtiss, G., Demery, J. A., Lebowitz, B. K. & Vanderploeg, R. D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society* **11**: 215-227.

Bigler, E. D. (2001). The lesion(s) in traumatic brain injury: Implications for clinical neuropsychology. *Archives of Clinical Neuropsychology* **16**(2): 95-131.

*Bohen, N., Jolles, J. & Twijnstra, A. (1992). Modification of the stroop colour-word test improves differentiation between patients with mild head injury and matched controls. *Clinical Neuropsychologist* **6**(2): 178-184.

*Bohnen, N. I., Jolles, J., Twijnstra, A., Mellink, R. & Wijnen, G. (1995). Late neurobehavioural symptoms after mild head injury. *Brain Injury* **9**(1): 27-34.

Bonne, O., Gilboa, A., Louzoun, Y., Kempf-Sherf, O., Katz, M., Fishman, Y., Ben-Nahum, Z., Krausz, Y., Bocher, M., Lester, H., Chisin, R. & Lerer, B. (2003). Cerebral blood flow in chronic symptomatic mild traumatic brain injury. *Psychiatry Research: Neuroimaging* **124**(3): 141-152.

Borg, J., Holm, L., casidy, J. D., Peloso, P. M., carroll, L. J., von Holst, H., Paniak, C. & Yates, D. (2004). Diagnostic procedures in mild traumatic brain injury: Results of the who collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine* **43S**: 61-75.

*Braun, C. M., Daigneault, S. & Champagne, D. (1989). Information processing deficits as indexed by reaction time parameters in severe closed head injury. *International Journal of Clinical Neuropsychology* **11**(4): 167-176.

Bush, G., Frazier, J. A., Rauch, S. L., Seidman, L. J., Whalen, P. J., Jenike, M. A., Rosen, B. R. & Biederman, J. (1999). Anterior cingulate cortex dysfunction in attention-

deficit/hyperactivity disorder revealed by fmri and the counting stroop. *Biological Psychiatry* **45**(12): 1542-1552.

Cabeza, R., Grady, C. L., Nyberg, L., McIntosh, A. R., Tulving, E., Kapur, S., Jennings, J. M., Houle, S. & Craik, F. I. M. (1997). Age-related differences in neural activity during memory encoding and retrieval: A positron emission tomography study. *J. Neurosci.* **17**(1): 391-400.

*Cantin, J. F., McFadyen, B. J., Doyon, J., Swaine, B., Dumas, D., Vallee, M., Cantin, J.-F., McFadyen, B. J., Doyon, J., Swaine, B., Dumas, D. & Vallee, M. (2007). Can measures of cognitive function predict locomotor behaviour in complex environments following a traumatic brain injury? *Brain Injury* **21**(3): 327-34.

Chambers, C. D., Bellgrove, M. A., Stokes, M. G., Henderson, T. R., Garavan, H., Robertson, I. H. & Mattingley, J. B. (2006). Executive 'brake failure' following deactivation of human frontal lobe. *Journal of Cognitive Neuroscience* **18**: 444-455.

Chambers, C. D., Garavan, H. & Bellgrove, M. A. (2009). Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neuroscience & Biobehavioral Reviews* **33**(5): 631-646.

Chan, R. C. (2005). Sustained attention in patients with mild traumatic brain injury. *Clinical Rehabilitation* **19**(2): 188-193.

*Chan, R. C. & Chan, R. C. K. (2005). Sustained attention in patients with mild traumatic brain injury. *Clinical Rehabilitation* **19**(2): 188-93.

Cohen, J. (1988). *Statistical power analysis for the behavioural sciences (2nd ed.)*. New Jersey, Lawrence Erlbaum.

Conners, C. K. (1995). *Conners' continuous performance test*. Toronto, Multi-Health Systems.

de Zubicaray, G. I., Andrew, C., Zelaya, F. O., Williams, S. C. R. & Dumanoir, C. (2000). Motor response suppression and the prepotent tendency to respond: A parametric fmri study. *Neuropsychologia* **38**(9): 1280-1291.

*De Haan, A., Halterman, C., Langan, J., Drew, A. S., Osternig, L. R., Chou, L. S., van Donkelaar, P., DeHaan, A., Halterman, C., Langan, J., Drew, A. S., Osternig, L. R., Chou, L.-S. & van Donkelaar, P. (2007). Cancelling planned actions following mild traumatic brain injury. *Neuropsychologia* **45**(2): 406-11.

Dimoska, A. & Johnstone, S. J. (2008). Effects of varying stop-signal probability on erps in the stop-signal task: Do they reflect variations in inhibitory processing or simply novelty effects? *Biological Psychology* **77**(3): 324-336.

*Dockree, P., Bellgrove, M., O'Keeffe, F., Moloney, P., Aimola, L., Carton, S. & Robertson, I. (2006). Sustained attention in traumatic brain injury (TBI) and healthy controls: Enhanced sensitivity with dual-task load. *Experimental Brain Research* **168**(1): 218-229.

*Dockree, P. M., Kelly, S. P., Roche, R. A. P., Hogan, M. J., Reilly, R. B. & Robertson, I. H. (2004). Behavioural and physiological impairments of sustained attention after traumatic brain injury. *Cognitive Brain Research* **20**(3): 403-414.

*Draper, K. & Ponsford, J. (2008). Cognitive functioning ten years following traumatic brain injury and rehabilitation. *Neuropsychology* **22**(5): 618-625.

Duncan, C. C., Kosmidis, M. H. & Mirsky, A. F. (2005). Closed head injury-related information processing deficits: An event-related potential analysis. *International Journal of Psychophysiology* **58**(2-3): 133-157.

Felmingham, K. L., Baguley, I. J. & Green, A. M. (2004). Effects of diffuse axonal injury on speed of information processing following severe traumatic brain injury. *Neuropsychology* **18**(3): 564-571.

*Felmingham, K. L., Baguley, I. J., Green, A. M., Felmingham, K. L., Baguley, I. J. & Green, A. M. (2004). Effects of diffuse axonal injury on speed of information processing following severe traumatic brain injury. *Neuropsychology* **18**(3): 564-71.

Frenchmen, K. A., Fox, A. M. & Mayberry, M. T. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. . *Journal of Clinical and Experimental Neuropsychology* **27**: 334-351.

*Gagnon, J., Bouchard, M.-A., Rainville, C., Lecours, S. & St-Amand, J. (2006). Inhibition and object relations in borderline personality traits after traumatic brain injury. *Brain Injury* **20**(1): 67 - 81.

*Hales, M. K. (1999). Cognitive disengagement, visual disengagement, and semantic priming: An analysis of attentional and automatic processes in traumatic brain injured and control groups. *Department of Educational Psychology*. Utah, University of Utah. Doctor of Philosophy.

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

Hedges, L. V. & Olkin, I. (1985). *Statistical methods for meta-analysis*. Orlando, FL, Academic Press.

Hunter, J. E. & Schmidt, F. L. (1990). *Methods of meta-analysis: Correcting error and bias in research findings*. Newbury Park, California, Sage.

*Incoccia, C., Formisano, R., Muscato, P., Reali, G. & Zoccolotti, P. (2004). Reaction and movement times in individuals with chronic traumatic brain injury with good motor recovery. *Cortex* **40**(1): 111-115.

Johnstone, S. J., Barry, R. J., Markovska, V., Dimoska, A. & Clarke, A. R. (2008). Erps and task performance during response inhibition and interference control in children with ad/hd. *International Journal of Psychophysiology* **69**(3): 195-195.

Johnstone, S. J., Pleffer, C. B., Barry, R. J., Clarke, A. R. & Smith, J. L. (2005). Development of inhibitory processing during the go/nogo task: A behavioral and event-related potential study of children and adults. *Journal of Psychophysiology* **19**(1): 11-23.

Kelly, C., Hester, R., Foxe, J. J., Shpaner, M. & Garavan, H. (2006). Flexible cognitive control: Effects of individual differences and brief practice on a complex cognitive task. *NeuroImage* **31**: 866-886.

Kenemans, J. L., Wieleman, J. S. T., Zeegers, M. & Verbaten, M. N. (1999). Caffein and stroop interference. *Pharmacology, Biochemistry and Behaviour* **63**: 589-598.

Khan, F., Baguley, I. J. & Cameron, I. D. (2003). 4: Rehabilitation after traumatic brain injury. *Rehabilitation Medicine* **178**: 290-295.

*Killam, C., Cautin, R. L. & Santucci, A. C. (2005). Assessing the enduring residual neuropsychological effects of head trauma in college athletes who participate in contact sports. *Archives of Clinical Neuropsychology* **20**(5): 599-611.

Lansbergen, M. M., Kenemans, J. L. & van Engeland, H. (2007). Stroop interference and attention-deficit/hyperactivity disorder: A review and meta-analysis. *Neuropsychology* **21**(2): 251-262.

Larson, M. J., Kaufman, D. A., Schmalfuss, I. M. & Perlstein, W. M. (2007). Performance monitoring, error processing, and evaluative control following severe tbi. *Journal of the International Neuropsychological Society* **13**(6): 961-971.

*Larson, M. J., Kaufman, D. A., Schmalfuss, I. M., Perlstein, W. M., Larson, M. J., Kaufman, D. A. S., Schmalfuss, I. M. & Perlstein, W. M. (2007). Performance

monitoring, error processing, and evaluative control following severe tbi. *Journal of the International Neuropsychological Society* **13**(6): 961-71.

Levin, H. & Kraus, M. F. (1994). The frontal lobes and traumatic brain injury. *J Neuropsychiatry Clin Neurosci* **6**(4): 443-454.

Logan, G. D. (1994). *On the ability to inhibit thought and action: A users' guide to the stop signal paradigm*. San Diego, CA, Academic Press.

Loken, W. J., Thornton, A. E., Otto, R. L. & Long, C. J. (1995). Sustained attention after severe closed head injury. *Neuropsychology* **9**(4): 592-598.

*Manly, T., Owen, A. M., McAvinue, L., Datta, A., Lewis, G. H., Scott, S. K., Rorden, C., Pickard, J. & Robertson, I. H. (2003). Enhancing the sensitivity of a sustained attention task to frontal damage: Convergent clinical and functional imaging evidence. *Neurocase: The Neural Basis of Cognition* **9**(4): 340 - 349.

Marsh, N. V. & Knight, R. G. (1991). Relationship between cognitive deficits and social skill after head injury. *Neuropsychology* **5**: 107-117.

Mathias, J. L. & Wheaton, P. (2007). Changes in attention and information-processing speed following severe traumatic brain injury: A meta-analytic review. *Neuropsychology* **21**(2): 212-223.

McAllister, T., Flashman, L., Harker Rhodes, C., Tyler, A., Moore, J., Saykin, A., McDonald, B., Tosteson, T. & Tsongalis, G. (2008). Single nucleotide polymorphisms in ankk1 and the dopamine d2 receptor gene affect cognitive outcome shortly after traumatic brain injury: A replication and extension study. *Brain Injury* **22**: 705-714.

McAllister, T. W., Saykin, A. J., Flashman, L. A., Sparling, M. B., Johnson, S. C., Guerin, S. J., Mamourian, A. C., Waeaver, J. B. & Yanofsky, N. (1999). Brain activation during working memory one month after mild traumatic brain injury: A functional mri study. *Neurology* **12**: 1300-1308.

McAllister, T. W., Sparling, M. B., Flashman, L. A., Guerin, S. J., Mamourian, A. C. & Saykin, A. J. (2001). Differential working memory load effects after mild traumatic brain injury. *NeuroImage* **14**(5): 1004-1012.

*McAvinue, L., O'Keeffe, F., McMackin, D. & Robertson, I. H. (2005). Impaired sustained attention and error awareness in traumatic brain injury: Implications for insight. *Neuropsychological Rehabilitation: An International Journal* **15**(5): 569 - 587.

McDonald, S. (2005). Are you laughing or crying? Deficits in emotion perception following severe traumatic brain injury. *Brain Impairment* **6**: 56-67.

Milich, R., Hartung, C. M., Martin, C. A. & Haigler, E. D. (1994). Behavioural disinhibition and underlying processes in adolescents with disruptive disorders. *Disruptive behaviour disorders in childhood*. D. K. Routh. New York, Erlbaum**:** 109-138.

Nee, D. E., Wager, T. D. & Jonides, J. (2007). Interference resolution: Insights from a meta-analysis of neuroimaging tasks. *Cognitive, Affective, & Behavioral Neuroscience* **7**(1): 1-17.

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin* **126**(2): 220-246.

*O'Keeffe, F. M., Dockree, P. M. & Robertson, I. H. (2004). Poor insight in traumatic brain injury mediated by impaired error processing?: Evidence from electrodermal activity. *Cognitive Brain Research* **22**(1): 101-112.

*O'Keeffe, F., Dockree, P., Moloney, P., Carton, S. & Robertson, I. (2007). Characterising error-awareness of attentional lapses and inhibitory control failures in patients with traumatic brain injury. *Experimental Brain Research* **180**(1): 59-67.

Park, D. C. (2000). The basic mechanisms accounting for age-related decline in cognitive function. *Cognitive aging: A primer*. D.C. Park &N. Schwarz. Philadelphia, Psychology Press**:** 3-22.

*Perlstein, W. M., Cole, M. A., Demery, J. A., Seignourel, P. J., Dixit, N. K., Larson, M. J. & Briggs, R. W. (2004). Parametric manipulation of working memory load in traumatic brain injury: Behavioral and neural correlates. *Journal of the International Neuropsychological Society* **10**(05): 724-741.

Perlstein, W. M., Larson, M. J., Dotson, V. M. & Kelly, K. G. (2006). Temporal dissociation of components of cognitive control dysfunction in severe tbi: Erps and the cued-stroop task. *Neuropsychologia* **44**(2): 260-274.

Ponsford, J. & Kinsella, G. (1992). Attention deficits following closed-head injury. *Journal of Clinical and Experimental Neuropsychology* **14**(5): 822-838.

*Ponsford, J. & Kinsella, G. (1992). Attentional deficits following closed-head injury: The role of inhibitory processes. *Journal of Clinical and Experimental Neuropsychology* **14**(822-838).

Ponsford, J. L. (2000). Attention. *Neuropsychological assessment in clinical practice: A guide to test interpretation and integration*. G. Groth-Marnat. Hoboken, NJ, John Wiley & Sons Inc**:** 355-400.

*Potter, D. D., Jory, S. H., Bassett, M. R., Barrett, K. & Mychalkiw, W. (2002). Effect of mild head injury on event-related potential correlates of stroop task performance. *Journal of the International Neuropsychological Society* **8**(6): 828-837.

Ramautar, J. R., Kok, A. & Ridderinkhof, K. R. (2004). Effects of stop-signal probability in the stop-signal paradigm: The n2/p3 complex further validated. *Brain and Cognition* **56**(2): 234-252.

Rao, V. & Lyketsos, C. (2000). Neuropsychiatric sequelae of traumatic brain injury. *Psychosomatics* **41**(2): 95-103.

Ray-Li, C.-S., Huang, C., Constable, R. T. & Sinha, R. (2006). Gender differences in the neural correlates of response inhibition during a stop-signal task. *Neuroimage* **32**(4): 1918-1929.

*Rieger, M. & Gauggel, S. (2002). Inhibition of ongoing responses in patients with traumatic brain injury. *Neuropsychologia* **40**(1): 76-85.

*Rios, M., Perianez, J. A. & Munoz-Cespedes, J. M. (2004). Attentional control and slowness of information processing after severe traumatic brain injury. *Brain Injury* **18**(3): 257-272.

*Robertson, I. H., Manly, T., Andrade, J., Baddeley, B. T. & Yiend, J. (1997). 'oops!': Performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia* **35**(6): 747-58.

*Roche, R. A., Dockree, P. M., Garavan, H., Foxe, J. J., Robertson, I. H., O'Mara, S. M., Roche, R. A. P., Dockree, P. M., Garavan, H., Foxe, J. J., Robertson, I. H. & O'Mara, S. M. (2004). Eeg alpha power changes reflect response inhibition deficits after traumatic brain injury (tbi) in humans. *Neuroscience Letters* **362**(1): 1-5.

Roche, R. A. P., Dockree, P. M., Garavan, H., Foxe, J. J., Robertson, I. H. & O'Mara, S. M. (2004). Eeg alpha power changes reflect response inhibition deficits after traumatic brain injury (tbi) in humans. *Neuroscience Letters* **362**(1): 1-5.

Rosenberg, M. S. (2005). The file-drawer problem revisited: A general weighted method for calculating fail-safe numbers in meta-analysis. *Evolution* **59**(2): 464-468.

Rubia, K., Russell, T., Overmeyer, S., Brammer, M. J., Bullmore, E. T., Sharma, T., Simmons, A., Williams, S. C. R., Giampietro, V., Andrew, C. M. & Taylor, E. (2001).

Mapping motor inhibition: Conjunctive brain activations across different versions of go/no-go and stop tasks. *Neuroimage* **13**(2): 250-261.

*Rugg, M. D., Cowan, C. P., Nagy, M. E., Milner, A. D., Jacobson, I. & Brooks, D. N. (1989). Cnv abnormalities following closed head injury. *Brain* **112**: 489-506.

Schretlen, D. J. & Shapiro, A. M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry* **15**(4): 341-349.

*Schroeter, M. L., Ettrich, B., Schwier, C., Scheid, R., Guthke, T., von Cramon, D. Y., Schroeter, M. L., Ettrich, B., Schwier, C., Scheid, R., Guthke, T. & von Cramon, D. Y. (2007). Diffuse axonal injury due to traumatic brain injury alters inhibition of imitative response tendencies. *Neuropsychologia* **45**(14): 3149-56.

*Seignourel, P. J., Robins, D. L., Larson, M. J., Demery, J. A., Cole, M. & Perlstein, W. M. (2005). Cognitive control in closed head injury: Context maintenance dysfunction or prepotent response inhibition deficit? *Neuropsychology* **19**(5): 578-590.

*Simpson, A. & Schmitter-Edgecombe, M. (2000). Intactness of inhibitory attentional mechanisms following severe closed-head injury. *Neuropsychology* **14**(2): 310-319.

Soeda, A., Nakashima, T., Okumura, A., Kuwata, K., Shinoda, J. & Iwama, T. (2005). Cognitive impairment after traumatic brain injury: A functional magnetic resonance imaging study using the stroop task. *Diagnostic Neuroradiology* **47**: 501-506.

Spikeman, A., Deelman, B. G. & van Zomeren, A. H. (2000). Executive functioning, attention and frontal lesions in patients with chronic chi. *Jounral of Clinical and Experimental Neuropsychology* **22**: 325-338.

*Spikeman, J. M., van Zomeren, A. H. & Deelman, B. G. (1996). Deficits of attention after closed-head injury: Slowness only? *Journal of Clinical and Experimental Neuropsychology* **18**(5): 755-767.

Starkstein, S. E. (1997). Mechanism of disinhibition after brain lesions. *Journal of Nervous & Mental Disease* **185**(2): 108-114.

*Stewart, J.A. L. & Tannock, R. (1999). Inhibitory control differences following mild head injury. *Brain and Cognition* **41**(3): 411-416.

Stuss, D. T., Floden, D., Alexander, M. P., Levine, B. & Katz, D. (2001). Stroop performance in focal lesion patients: Dissociation of processes and frontal lobe lesion location. *Neuropsychologia* **39**(8): 771-786.

Stuss, D. T., Hugenholtz, H., Richard, M., LaRochelle, S., Poirier, C. A. & Bell, I. (1985). Subtle neuropsychological deficits in patients with good recovery after closed head injury. *neurosurgery* **17**: 41-47.

*Summers, M. J. & Summers, M. J. (2006). Increased inattentional blindness in severe traumatic brain injury: Evidence for reduced distractibility? *Brain Injury* **20**(1): 51-60.

Tate, R. L. (1999). Executive dysfunction and characterological changes after traumatic brain injury: Two sides of the same coin? *Cortex* **35**(1): 39-55.

Tate, R. L., McDonald, S. & Lulham, J. M. (1998). Incidence of hospital-treated traumatic brain injury in an australian community. *Australian and New Zealand Journal of Public Health* **22**: 419-423.

*Vakil, E., Weisz, H., Jedwab, L., Groswasser, Z. & Aberbuch, S. (1995). Stroop color-word task as a measure of selective attention: Efficiency in closed-head-injured patients. *Journal of Clinical and Experimental Neuropsychology* **17**(3): 335 - 342.

van den Wildenberg, W. P. M. & van der Molen, M. W. (2004). Developmental trends in simple and selective inhibition of compatible and incompatible responses. *Journal of Experimental Child Psychology* **87**(3): 201-220.

van Mourik, R., Oosterlaan , J. & Sergeant, J. A. (2005). The stroop revisited: A meta-analysis of interference control in ad/hd. *Journal of Child Psychology and Psychiatry* **46**(2): 150-165.

Verbruggen, F., Liefooghe, B., Notebaert, W. & Vandierendonck, A. (2005). Effects of stimulus-stimulus compatibility and stimulus-response compatibility on response inhibition. *Acta Psychologica* **120**(3): 307-326.

Wager, T. D., Sylvester, C.-Y. C., Lacey, S. C., Nee, D. E., Franklin, M. & Jonides, J. (2005). Common and unique components of response inhibition revealed by fmri. *Neuroimage* **27**(2): 323-340.

*Whyte, J., Grieb-Neff, P., Gantz, C., Polansky, M., Whyte, J., Grieb-Neff, P., Gantz, C. & Polansky, M. (2006). Measuring sustained attention after traumatic brain injury: Differences in key findings from the sustained attention to response task (sart). *Neuropsychologia* **44**(10): 2007-14.

Zakzanis, K., Leach, L. & Kaplan, E. (1999). *Neuropsychological differential diagnosis*. Lisse Netherlands, Swets & Zeitlinger.

Zakzanis, K. K. (2001). Statistics to tell the truth, the whole truth, and nothing but the truth: Formulae, illustrative numerical examples, and heuristic interpretation of effect size analyses for neuropsychological researchers. *Archives of Clinical Neuropsychology* **16**(7): 653-667.