Published in final edited form as:

J Clin Exp Neuropsychol. 2011 August ; 33(7): 805–819. doi:10.1080/13803395.2011.562864.

Traumatic Brain Injury and Secondary Attention-Deficit/ Hyperactivity Disorder in Children and Adolescents: The Effect of Reward on Inhibitory Control

Katia J. Sinopoli^{a,d,e}, Russell Schachar^{b,c,e}, and Maureen Dennis^{b,e,f}

^aPhysiology and Experimental Medicine, The Hospital for Sick Children, 555 University Ave., Toronto, Ontario, Canada, M5G 1X8

^bNeurosciences and Mental Health, The Hospital for Sick Children, 555 University Ave., Toronto, Ontario, Canada, M5G 1X8

^cPsychiatry Department, The Hospital for Sick Children, 555 University Ave., Toronto, Ontario, Canada, M5G 1X8

^dPsychology Department, The Hospital for Sick Children, 555 University Ave., Toronto, Ontario, Canada, M5G 1X8

^eFaculty of Medicine, University of Toronto, 500 University Ave., Toronto, Ontario, Canada, M5G 1V7

^fDepartment of Psychology, University of Toronto, 500 University Ave., Toronto, Ontario, Canada, M5G 1V7

Abstract

Poor inhibitory control and abnormalities in responding to rewards are characteristic of the developmental or primary form of attention-deficit/hyperactivity disorder (P-ADHD). A secondary form of ADHD (S-ADHD) may occur as a consequence of childhood traumatic brain injury (TBI), but the similarities and differences between these two forms of ADHD have not been well characterized. To address these issues, we studied two inhibitory control tasks under different reward conditions in four groups of children and adolescents: TBI who did not exhibit S-ADHD, TBI who did exhibit S-ADHD, P-ADHD, and healthy controls. Participants with TBI exhibited poor cancellation inhibition relative to controls. Although reward facilitated both cancellation and restraint inhibition similarly across groups, poor performance persisted in the P-ADHD group, and participants with S-ADHD exhibited a selective deficit in cancellation inhibition.

Keywords

TBI; ADHD; inhibitory control; rewards; development

Traumatic brain injury (TBI) is common in children and adolescents, and often results in significant cognitive and behavioral deficits, including inattention (Catroppa, Anderson, & Stargatt, 1999; Cicerone, 1996; Dennis, Wilkinson, Koski, & Humphreys, 1995) and poor inhibitory control (Levin, Hanten, Zhang, Swank, Hunter, 2004; Levin et al., 1993; Konrad, Gauggel, Manz, & Scholl, 2000a, 2000b). Childhood TBI may also lead to the emergence of

new-onset psychiatric disorders such as attention-deficit/hyperactivity disorder (i.e., secondary ADHD (S-ADHD); Gerring et al., 1998). S-ADHD occurs in approximately 15–20% of TBI survivors without a previous history of ADHD (Gerring et al., 1998; Gerring et al., 2000; Herskovits et al., 1999; Max et al., 2005a, 2005b; Slomine et al., 2005; Yeates et al., 2005), manifesting as early as 6 months post-TBI (Max et al., 2005a) and persisting into the chronic stage of recovery (e.g., Max et al., 1998; Max et al., 2005b).

A key question is whether S-ADHD is simply an acquired form of developmental or primary ADHD (P-ADHD), sharing commonalities in cognitive, behavioral and functional outcomes, or whether fundamental differences exist between these disorders despite similarities in some overt behavioral features. Both P-ADHD and S-ADHD exhibit deficits with inhibitory control (e.g., Schachar, Levin, Max, Purvis, & Chen, 2004; Schachar et al., 2007) but it is not clear that the inhibitory control deficits are identical in the two groups.

An important distinction in inhibitory control tasks is that between canceling or stopping an ongoing prepotent response and restraining or withholding an action. Cancellation is often measured with the stop signal task (Logan & Cowan, 1984; Logan, 1994), in which participants are instructed to respond as quickly as possible to one of 2 visual “go” signals and stop responding when they hear the auditory stop signal (which is presented after varying delays following the go signal). Poor or inefficient inhibition is manifested as longer latencies to respond to the stop signal, or slower stop signal reaction time (SSRT). Restraint is commonly measured with the go/no-go (GNG) task which requires participants to respond as quickly as possible to a visual go stimulus, but to withhold their responses when they encounter a visual “no-go” stimulus. Poor restraint is indexed by the number of responses made to no-go stimuli (commission errors). Children with P-ADHD often exhibit deficits in both cancellation and restraint (e.g., Schachar, Levin, et al., 2004; Schachar et al., 2007). The close association between the cognitive and behavioral aspects of inhibitory control suggests that poor inhibition may represent a vulnerability to ADHD (Young et al., 2009).

In addition to their deficits in inhibitory control, children with P-ADHD also exhibit abnormal responses to rewards, with some theorists speculating that these children have a preference for immediate reward (Sonuga-Barke, 2002) and/or an insensitivity to reward whereby that more reinforcement is required to motivate behavior (e.g. Haenlein and Caul, 1987). To date, the effects of reward on inhibitory control in children with P-ADHD are equivocal, with reports of reward facilitation (Desman, Petermann & Hampel, 2008; Huang-Pollock, Mikami, Pfiffner, & McBurnett, 2007; Konrad et al., 2000b; Scheres, Oosterlaan, & Sergeant, 2001; Stevens, Quittner, Zuckerman, & Moore, 2002; Tamm & Carlson, 2007), reward impairment (Gomez, 2003; Shanahan, Pennington, & Willcutt, 2008; Wodka et al. 2007), or null effects (Crone, Jennings, & van der Molen, 2003; Iaboni, Douglas, & Baker, 1995; Oosterlaan & Sergeant, 1998). Methodological problems that complicate interpretation within and across these studies include: lack of a control group (Tamm & Carlson, 2007); no non-reward condition to compare reinforcement effects to (Huang-Pollock et al., 2007; Iaboni et al., 1995; Oosterlaan & Sergeant, 1998); confounding of rewards and punishments (Crone et al., 2003; Gomez, 2003; Shanahan et al., 2008; Wodka et al., 2007); rewarding both response execution and inhibition (Huang-Pollock et al., 2007; Shanahan et al., 2008; Stevens et al., 2002); the finding that feedback alone produces similar improvements to rewards (Desman et al., 2008); and the existence of normal inhibition in P-ADHD groups (Crone et al., 2003; Scheres et al., 2001), which makes it difficult to ascertain if rewards facilitated or impaired performance.

The nature and magnitude of inhibitory control difficulties in children with S-ADHD have not been well-studied, and it is currently unknown whether P-ADHD and S-ADHD exhibit similar deficits across all inhibitory control tasks. Relative to typically developing children,

children with S-ADHD exhibit poor cancellation inhibition on the stop signal task (Konrad et al., 2000a, 2000b). An inhibition deficit has been documented relative to children with TBI in those with *both* a severe TBI and S-ADHD diagnosis (Schachar, Chen, et al., 2004), suggesting that those with greater injury severity and a *de novo* diagnosis of ADHD are more susceptible to disinhibition than those with less severe injuries. Because most studies include children in both chronic and acute phases of recovery, the interpretation of data on inhibition in TBI and S-ADHD populations is not clear. For instance, children with TBI exhibit an initial deficit in SSRT that resolves by two years post-injury (Leblanc et al., 2005). Further, commission errors on the GNG task decrease with time since injury, with greater injury severity resulting in poorer improvement over time (Wassenberg, Max, Lindgren, & Schatz, 2004). To date, only two studies have assessed cancellation performance in both P-ADHD and S-ADHD groups (Konrad et al., 2000a, 2000b), but because the performance was not directly compared between groups, it is currently unknown whether or not these patients cancel their responses in a similar manner. No study has compared the performance of children with P-ADHD and S-ADHD on other forms of inhibitory control, such as restraint.

Little is known about how reward variables influences inhibition in children with S-ADHD. Konrad and colleagues (2000b) rewarded successful inhibition on the stop signal task, which improved cancellation performance in children with TBI and S-ADHD, although not to the point of comparability to controls. At the same time, reward facilitated inhibition in children with P-ADHD such that their performance was similar to controls. However, the ADHD groups were not directly compared in this study (Konrad et al., 2000b), so it is unknown whether or not they responded similarly to reward.

While comparisons to non-injured controls are helpful in determining whether and how children with S-ADHD differ from typically functioning children in terms of inhibitory control and responses to reward, and comparisons to children with TBI without S-ADHD indicate the specific deficits S-ADHD confers beyond the consequences of TBI, a direct comparison to children with P-ADHD may reveal the degree and intensity of deficits in S-ADHD. This later comparison would also clarify whether each form of ADHD reflects a similar disinhibition syndrome, despite differences in etiology.

Although childhood TBI can affect a number of cognitive processes, poor inhibitory control on the stop signal task (longer SSRT) has been proposed as a candidate endophenotype for P-ADHD (Crosbie, Perusse, Barr, & Schachar, 2008). If children with TBI exhibit similar inhibition deficits to those with ADHD, then one would need to qualify the relationship between endophenotype and genetic risk. If children with S-ADHD exhibit similar deficits in cancellation and restraint to children with P-ADHD, then S-ADHD may be considered as an acquired disorder that mimics the P-ADHD endophenotype, which would inform not only the brain bases of inhibition deficits in P-ADHD, but also the neural bases of inhibitory control in general. If the two groups differ across measures or exhibit a dissimilar pattern of results, then there is evidence that S-ADHD is a separate disorder from P-ADHD, in terms of both underlying mechanisms and associated behavioral deficits. If both groups of children respond similarly to reward despite differences in inhibitory control performance, then it may be concluded that rewards operate similarly on inhibition abilities across groups suggesting a powerful effect of motivational variables in spite of differences in brain abnormalities. In short, each pattern of results would have specific and direct implications for the assessment and treatment of S-ADHD.

The objective of the present study was to understand how reward modulates cancellation and restraint inhibition in developmental (P-ADHD) and acquired (S-ADHD) forms of ADHD. We compared restraint and cancellation inhibitory control in four groups of participants

(typically developing children, children with P-ADHD, children with TBI in the chronic stage of recovery, and children with TBI in the chronic stage of recovery with a research diagnosis of S-ADHD). To measure cancellation and restraint we used two versions of the stop signal task that differed only in the timing of the presentation of the stop signal (Schachar et al., 2007). By making the no-go signal in the restraint version of the stop signal task an auditory tone, task demands in restraint and cancellation tasks are equated, thereby minimizing reliance on selective attention that are associated with the traditional GNG task (Aron & Poldrack, 2005; Rubia, Smith, Brammer, & Taylor, 2003). Following the administration of the standard inhibitory control tasks, we then investigated the relation between reward and inhibitory control by rewarding successful cancellation and restraint.

Predictions concerned both group and reward effects, and were aligned with the literature reviewed earlier. We predicted that approximately 15–20 % of children with TBI would exhibit symptoms consistent with a S-ADHD diagnosis, and these participants were expected to exhibit poorer inhibitory control on measures of cancellation and restraint relative to those with TBI only or to healthy controls. Given that time since injury ameliorates cancellation deficits in children with TBI (Leblanc et al., 2005), and that participants in the TBI group in the present study were also in the chronic phase of recovery, we predicted that children with TBI without S-ADHD would exhibit inhibitory control similar to that of normal controls under all reward conditions. Participants in the P-ADHD group were expected to have poorer inhibitory control relative to controls, with rewards possibly improving inhibitory control performance. Improvement of inhibitory control deficits by rewards in the P-ADHD group to levels exhibited by typically developing children would suggest that children with P-ADHD also suffer from an inability to self-motivate their own behavior.

Method

Recruitment

Children and adolescents aged seven to 17 years old were recruited to form four participant groups: Control, TBI, S-ADHD, and P-ADHD groups. Participants in the TBI groups (n=63) were recruited through trauma registries at the Hospital for Sick Children in Toronto. Typically developing youths (Control group, n=66) and children and adolescents with diagnosed P-ADHD (n=20) were recruited through community advertisements. A brief recruitment phone interview with each participant's parent collected demographic information and ensured participation eligibility: Control participants were excluded if their parents reported that they had a history of P-ADHD, TBI, or learning disabilities; youths in the TBI and S-ADHD groups were excluded if there was evidence of pre-injury P-ADHD and a history of more than one TBI; exclusion for the P-ADHD group included a history of TBI. For all groups, participants were excluded if there was a history of neurological disorders, severe psychiatric disorders (e.g., psychosis), mental retardation, and/or sensory or motor impairments. Family socioeconomic (SES) scores were calculated from the Hollingshead Four-Factor Index (Hollingshead, 1975) by averaging the SES scores of both parents, or from one parent, when this was the only information available.

The Conners 3rd Edition Rating Scales

The full-length form of the Conners 3rd Edition Rating Scales (Conners 3; Conners, 2008) was administered to each participant's parent (110 items) and teacher (115 items). The Conners 3 assesses symptoms of ADHD and associated behavioral difficulties, such as learning problems, executive dysfunction, and aggression, as well as symptoms related to common comorbidities of ADHD such as oppositional defiant disorder and conduct disorder. The Conners 3 also includes symptom counts, which are explicit Diagnostic and

Statistical Manual, 4th edition text revision (DSM-IV-TR; American Psychiatric Association, 2000) criteria that are necessary to diagnose ADHD, oppositional defiant disorder, and conduct disorder. Thus, this rating scale was used to confirm inclusion of participants into each group and to identify children with TBI who also reached criteria for S-ADHD.

Participants were excluded from the Control group if they reached criteria for any disorder on the DSM-IV-TR Symptom Counts and/or if they were rated as exhibiting a T-score in the atypical range (i.e., over 65) on any subscale of the Conners 3 parent or teacher scales. Participants in the P-ADHD group were excluded if they failed to reach DSM-IV-TR criteria for ADHD (any subtype) on *both* the Conners 3 parent and teacher scales. Because age at onset of S-ADHD can be no earlier than the age at TBI, we waived the age requirement for diagnosis as well as the requirement that ADHD symptoms must be present in at least two environments (i.e., since it is not clear that symptoms need to be present in multiple environments to diagnose S-ADHD; American Psychiatric Association, 2000). Therefore, participants in the TBI group were included into the S-ADHD group if they reached DSM-IV-TR criteria for ADHD (any subtype) on *either* the Conners 3 parent *or* teacher scale.

Participants

Control Group—Of the 66 youths initially recruited into the Control group, nine participants cancelled or missed their appointments and 13 participants were excluded based on clinically significant elevations on the Conners 3 rating scales. The final Control group consisted of 44 participants (21 female, 23 male; mean age, 12.44 years).

TBI and S-ADHD groups—Inclusion criteria included the presence of a non-inflicted, non-penetrating TBI at least one year but no more than six years prior to testing, hospitalization for at least one night for the TBI, the absence of multiple head injuries, and no pre-injury neurological disorders. Ten participants cancelled or missed their appointments, and four participants were excluded due to the presence of a pre-injury P-ADHD diagnosis. Nine participants without evidence of pre-injury P-ADHD met criteria for ADHD (any subtype) on parent and/or teacher Conners 3 ratings of DSM-IV-TR ADHD symptoms, and were included into the S-ADHD group (5 female, 4 male; mean age, 11.91 years). While the participant number of the S-ADHD group is relatively small, it does represent the incidence of S-ADHD following childhood TBI (see Gerring et al., 1998; Max et al., 2005a, 2005b). Forty participants without evidence of ADHD on the Conners 3 remained in the TBI group (16 female, 24 male; mean age, 11.8 years).

Severity of TBI was classified based on initial post-resuscitation Glasgow Coma Scale (GCS) scores (Teasdale & Jennet, 1974). For patients who did not require resuscitation, the lowest GCS score on record was used. GCS scores separated participants into 3 severity classifications: mild (TBI, n=28; S-ADHD, n=6), moderate (TBI, n=6; S-ADHD, n=2), and severe (TBI, n=16; S-ADHD, n=1) injuries.

P-ADHD—Inclusion criteria included the diagnosis of ADHD from a health care professional and a negative history for TBI. Two participants were excluded from further analyses due to a lack of significant elevations on Conners 3, and 1 participant initially recruited for the control group was included in the P-ADHD group based on significant elevations on the same scale. The final P-ADHD group (n=19; 2 female, 17 male; mean age, 10.70 years) included 12 participants who were either drug naïve or were not currently taking any psychotropic medication for their ADHD. The remaining participants were currently undergoing pharmacological treatment (with two participants taking multiple

medications); these participants were asked to discontinue treatment for 24 hours prior to testing.

Intelligence Testing

The Wechsler Abbreviated Scale of Intelligence (WASI) was administered to all participants. WASI scores were used to exclude any participant with a Full Scale IQ score under 75.

Stop Signal Tasks

The stop signal task is based on a formal model that conceptualizes the ability to stop a speeded motor response as the outcome of a race between response execution (“go” processes) and response inhibition (“stop” processes; Logan & Cowan, 1984; Logan, 1994; Verbruggen & Logan, 2008a); whichever process wins the race determines whether the response will be executed or inhibited. The race model allows for the measurement of the latency of inhibitory control (i.e., the SSRT), which is an unobservable, internally generated act of control (Logan, 1994). Two versions of the stop signal task were used to measure canceling (“cancellation”) and restraining (“restraint”) a prepotent response (see Figure 1; Logan, 1994; Schachar et al., 2007). Both versions consisted of a primary visual choice reaction time task involving a *go signal* and a secondary task involving a *stop signal* (Logan & Cowan, 1984; Logan, 1994). In the primary task, one of two possible go signals (an X or an O) was presented on a computer screen for 1000 milliseconds following a 500 millisecond fixation point. Participants were instructed to respond to these stimuli as quickly as possible by making the appropriate button press on a handheld controller. They were also instructed to inhibit their responses to the go signal when they heard the auditory stop signal (a 1000 Hz tone emitted from computer speakers). The stop signal occurred randomly on 25% of trials.

In the cancellation version, the stop signal occurred at various delays following the go signal to interrupt an already executed response. The delay between the presentation of the go and the stop signal was varied dynamically in this version of the task to ensure that participants would cancel their responses on 50% of the inhibition trials, regardless of whether or not they slowed their go reaction time as a strategy to increase successful cancellation (Logan & Cowan, 1984; Logan, 1994). The initial stop signal delay was set at 250 milliseconds. If a participant successfully inhibited his/her response, then the delay was increased by 50 milliseconds on the subsequent stop trial, and if the participant was unable to inhibit his/her response, then the delay was decreased by 50 milliseconds on the subsequent stop trial. This method ensured approximate control over stop and go processes by increasing or decreasing the ease of successful inhibition. In the restraint version of the task, the stop signal was presented concurrently with the go signal to prevent a response from being executed (Schachar et al., 2007). Thus, the delay between the presentation of the go and stop signals in the restraint task was always zero.

The latency of inhibitory control on both versions of the stop signal task was estimated by determining the SSRT via the integration procedure (Logan, 1994): the go reaction time in non-stop signal trials were rank ordered, the go reaction time that corresponded to the probability of inhibition was determined, and the SSRT was estimated by subtracting the mean delay from the new “integrated go reaction time”. By utilizing this procedure, all faster go reaction times that would have been executed and all slower responses that would have been stopped can be excluded from the final SSRT metric (Schachar et al., 2007).

Each task version was performed under three different reward conditions presented in the same ascending order of magnitude (NO, LOW, HIGH). In the neutral (NO) condition, the

words “Good work! Press a button to keep going” appeared in the middle of the screen. No other specific feedback about task performance was revealed. Following the NO condition, participants were informed that they would be performing the task for a second and third time where they could win points for successful inhibition, and if they could win 400 points by the end of the testing session, they would receive a prize (a \$10 gift certificate for the movies). They were also told that each time that they successfully inhibited their responses they would win 2 points in the low reward condition (LOW), which would be followed by a high reward condition (HIGH) where they would win 10 points. Points earned were immediately represented on the screen in a visual analog scale (the scale was left empty in the NO condition). Participants were informed of how many points they had won in each block via a message in the middle of the screen (e.g. “You won 12 points.”). Participants were also told that they would receive bonus points for pushing the buttons as quickly as possible during the primary task. Although no actual bonus points were rewarded, this additional instruction was given in order to reduce the tendency for participants to slow their go reaction time to ensure successful inhibition, which would have invalidated the race model (Logan, 1994). The points were recorded by the examiner, and total scores were revealed at the end of the testing session. When 2 children did not win enough points for the prize, their scores were bumped up to 400 via “bonus points” to ensure that all children won the prize.

Each reward condition of the cancellation and restraint versions of the SST involved a total of 120 trials divided into 5 blocks of 24 trials each, with the stop signal randomly occurring on 30 trials. The first block in each task was a practice block, which was not utilized in subsequent analyses. The reward conditions were always presented in the same ascending order of magnitude (NO, LOW, HIGH) due to a concern that the LOW or NO conditions could have been perceived as punishments if they followed presentation of the HIGH condition, which may have confounded the effects of rewards on inhibitory control with the effects of punishments, thus altering the main experimental manipulation.

The order of administration of each task version was counterbalanced, with the WASI and another computerized task intervening.

Procedure

Informed consent was obtained prior to testing from each participant’s parent, and assent was obtained from the participants themselves. All participants completed the WASI and both stop signal tasks, with order of task administration being randomized across participants. This experiment was conducted with approval of the Research Ethics Boards at the Hospital for Sick Children and the University of Toronto.

Statistical Analyses

Participant data from the stop signal task was excluded from statistical analyses if any of the following occurred: less than 66% accuracy on go trials, mean go reaction time (MRT) less than 100 milliseconds, and for the cancellation version only, percent inhibition less than 12.5% or greater than 87.5 % (See Band, 1997; Band, van der Molen, & Logan, 2003; Schachar, Levin, et al., 2004b; Verbruggen & Logan, 2008b). Based on these criteria, one participant from the Control group, two from the TBI group, and one participant from the P-ADHD group were excluded from analyses of the cancellation data, while one participant from the Control group and one participant from the P-ADHD group were excluded from analyses of the restraint data.

We examined both inhibitory control and response execution. Inhibitory control was indexed by SSRT and by the percentage of responses inhibited (percent inhibition). Percent

inhibition can be considered a more appropriate measure of restraint inhibition compared to the SSRT metric, as it provides information on the number of commission errors made on the task (i.e., a greater percentage of responses inhibited means less commission errors). Response execution was indexed by MRT, standard deviation of go reaction time (SDRT), and percent correct on go trials (accuracy). SDRT allowed for a measurement of variability, or the degree to which go responses were consistent across the tasks and reward conditions.

Age at injury, time since injury (for the TBI and S-ADHD groups only), age of assessment, WASI scores, and SES were compared between groups using an analysis of variance (ANOVA). These variables were also analyzed between severity groups (MILD, MODERATE, SEVERE) using a similar procedure.

Group differences in performance on each task were compared separately for each dependent variable (SSRT, percent inhibition, MRT, SDRT, and percent correct) utilizing repeated measures ANOVAs, with reward condition (NO, LOW, HIGH) and task (cancellation, restraint) as the within-subjects factors, and group (Control, TBI, S-ADHD, P-ADHD) and order of task administration (cancellation administered first, cancellation administered second) as between-subjects variables. Because inhibitory control improves with age (e.g., Williams, Ponesse, Schachar, Logan, & Tannock, 1999), age at test was added as a covariate in these analyses. We found significant differences between groups in terms of SES (see Results), thus SES was also included as a covariate. The effect of TBI severity on task performance was examined to ensure that injury-related variables were not mediating any group or task effects. Thus, similar repeated measures ANOVAs were conducted for each dependent variable with severity group (MILD, MODERATE, SEVERE) as the between-subjects variable.

Linear regression analyses determined the impact of demographic and injury variables on performance in the TBI and S-ADHD groups. GCS score, age at injury, time since injury, and SES score were entered as potential predictors of inhibitory control performance.

Results

Participant Characteristics

Participant characteristics are presented in Table 1. Age at assessment was similar between groups, and age of injury and time since injury did not differ between the TBI and S-ADHD groups. Age at injury, time since injury, IQ scores, and SES did not differ between severity groups. Although WASI scores differed between participant groups ($F(3, 107)=4.244$, $p=0.007$, partial $\eta^2=0.106$), mean IQ scores were within the normal range for all groups and no participants were excluded based on low IQ scores. The Control group exhibited higher SES scores relative to the other groups ($F(3, 102)=6.797$, $p<0.001$, partial $\eta^2=0.167$), thus SES was used as a covariate in subsequent analyses.

Stop Signal Tasks: Group and Reward Effects

SSRT—Participants in the Control group exhibited faster SSRT ($F(3, 92)=3.374$, $p=0.022$, partial $\eta^2=0.099$) relative to the TBI ($p=0.032$) and P-ADHD ($p=0.003$) groups (see Table 2). We found a reward by task interaction (linear effect, $F(2, 184)=3.182$, $p=0.044$, partial $\eta^2=0.033$): in the cancellation task, increasing reward magnitude decreased SSRT in a linear fashion (NO vs. LOW vs. HIGH); in the restraint task, improvements in SSRT were observed in the reward conditions (LOW and HIGH) compared to the neutral condition (NO), but no differences were found between the reward conditions themselves. SSRT was faster in the cancellation compared to the restraint task (linear effect, $F(1, 92)=17.860$,

$p < 0.001$, partial $\eta^2 = 0.163$). There was no significant effect of order, nor were there any other significant interactions.

Percent Inhibition—In the cancellation task, percent inhibition was approximately 50% across reward conditions and groups, indicating that the tracking algorithm was successful at limiting the probability of inhibition to approximately 0.5 (see Table 2). There was no main effect of group, but planned comparisons revealed a marginal difference between the P-ADHD group compared to the Control group ($p = 0.058$), with the P-ADHD group inhibiting a smaller percentage of responses. There was no main effect of reward, although there was a reward by task by order interaction (linear effect, $F(2, 184) = 6.475$, $p = 0.002$, partial $\eta^2 = 0.066$) revealing that if the cancellation task was administered first, participants inhibited a greater percentage of responses in the NO and LOW conditions of the cancellation task. As expected, a greater percentage of responses were inhibited in restraint compared to cancellation (linear effect, $F(1, 92) = 16.837$, $p < 0.001$, partial $\eta^2 = 0.155$). There were no other significant interactions.

MRT—There were no main effects of group, reward, task, or order of task presentation on MRT. There was a significant task by order interaction (linear effect, $F(1, 92) = 4.605$, $p = 0.035$, partial $\eta^2 = 0.048$; see Table 3), revealing that longer MRT was observed in the cancellation task when it administered first. There were no other significant interactions.

SDRT—Participants in the Control group exhibited less variability of go responses ($F(3, 92) = 3.575$, $p = 0.017$, partial $\eta^2 = 0.104$) compared to all other groups (TBI, $p = 0.027$; S-ADHD, $p = 0.038$; P-ADHD, $p = 0.005$; see Table 3). SDRT was not affected by reward. When the cancellation task was administered first, there was greater variability in the restraint task (task by order interaction; linear effect, $F(1, 92) = 10.016$, $p = 0.002$, partial $\eta^2 = 0.098$). There were no other significant interactions.

Percent Correct—There was a main effect of group ($F(3, 92) = 4.895$, $p = 0.003$, partial $\eta^2 = 0.138$), with greater accuracy on the go task observed in the Control ($p < 0.001$) and TBI ($p = 0.002$) groups relative to the P-ADHD group (see Table 3). Reward did not affect the accuracy of responses. There was, however, a task by order interaction (linear effect, $F(1, 92) = 22.280$, $p < 0.001$, partial $\eta^2 = 0.195$) and a reward by task by order interaction (linear effect, $F(2, 184) = 4.770$, $p = 0.010$, partial $\eta^2 = 0.049$). When the cancellation task was administered first, a greater percentage of correct go responses on the cancellation task were observed across all three reward conditions. In contrast, there was poorer accuracy on the restraint task when the cancellation task was administered first, especially in the NO and LOW conditions. There were no other significant interactions.

Injury Characteristics

Inhibitory Control Performance—There was no main effect of severity group on SSRT, although there was a significant reward by severity group interaction (quadratic effect, $F(4, 78) = 2.877$, $p = 0.028$, partial $\eta^2 = 0.129$) and a reward by task by severity group interaction (quadratic effect, $F(4, 78) = 3.513$, $p = 0.011$, partial $\eta^2 = 0.153$): faster SSRT in the MILD group was observed during the HIGH condition relative to the NO condition, especially during the cancellation task; and faster SSRT in the SEVERE group was observed during the reward (LOW and HIGH) conditions relative to the neutral (NO) condition, especially during the cancellation task. There was no effect of severity on percent inhibition.

“Go” Task Performance—There was no main effect of severity group on MRT, although there was a significant reward by task by group by order interaction (linear effect, $F(4, 78) = 4.696$, $p = 0.002$, partial $\eta^2 = 0.194$). When the cancellation task was administered

first, participants in the MILD group exhibited faster MRT during the cancellation task in the NO condition, while participants in the SEVERE group exhibited faster MRT in the NO condition of the cancellation task and faster MRT in the HIGH condition of the restraint task. The MODERATE severity group exhibited more variable go task performance ($F(2, 39)=5.624$, $p=0.007$, partial $\eta^2=0.224$), especially during the cancellation task (task by group interaction; linear effect, $F(2, 30)=3.895$, $p=0.029$, partial $\eta^2=0.166$) and when the cancellation task was administered second (task by group by order interaction; linear effect, $F(2, 78)=5.160$, $p=0.008$, partial $\eta^2=0.117$). Participants in the SEVERE group exhibited better accuracy during the NO condition relative to the HIGH condition (reward by severity interaction; linear effect, $F(4, 78)=2.486$, $p=0.05$, partial $\eta^2=0.113$), but poorer accuracy across all conditions when the cancellation task was administered first (reward by severity by order interaction; linear effect, $F(4, 78)=2.443$, $p=0.054$, partial $\eta^2=0.111$).

Regression Results: Cancellation—Age of injury significantly predicted MRT and SDRT across reward conditions (see Table 4); younger age at injury resulted in slower and more variable response execution in the neutral and reward conditions. A longer time since injury was also associated with faster MRT in the NO and LOW conditions. Age of injury and GCS score significantly predicted SSRT, but only in the HIGH condition (younger age of injury and lower GCS scores, or more severe injuries, resulted in longer SSRT). Injury variables were not significant predictors of accuracy on go trials or percent inhibition.

Regression Results: Restraint—Time since injury predicted MRT across reward conditions (greater time since injury, faster response execution). Age of injury was a significant predictor of MRT and SDRT performance across reward conditions (see Table 4), indicating that a younger age of injury results in slower and more variable response execution. For the LOW condition only, GCS score was also a significant predictor of MRT and SDRT, with participants with lower GCS scores (more severe injuries) exhibiting longer and more variable response execution time on the go task. Age at injury predicted SSRT in the LOW and HIGH conditions only, with longer SSRT observed in participants who had incurred their TBIs at younger ages. Accuracy in the HIGH condition was also significantly predicted by age at injury and time since injury, and individuals with an older age at injury and a longer time since injury were more accurate on the go task. Injury variables did not predict percent inhibition.

Discussion

The broadest objective of the present study was to investigate how reward affects cancellation and restraint inhibition under comparable task demands in developmental (P-ADHD) and acquired (S-ADHD) forms of attention disorders. In examining rewarded or unrewarded cancellation and restraint in children and adolescents with TBI, S-ADHD, P-ADHD, and typically developing controls, we aimed to delineate the inhibitory control profile of the four groups. Consistent with previous research (e.g. Schachar et al., 2007; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), participants in the P-ADHD group exhibited longer latencies to cancel and restrain their responses, inhibited fewer responses on the restraint version, produced more variable responses to go stimuli, and were less accurate in the cancellation go task relative to typically developing controls. In line our hypotheses and with previous research (e.g., Max et al., 2005b), 18 % of participants in the TBI group reached criteria for S-ADHD. This group of participants exhibited abnormal cancellation inhibition, and greater variability in go responses. Injury-related variables, such as age at injury, time since injury, and severity of injury did not mediate these group effects; rather, these variables were either unrelated or inconsistently related to inhibitory control. Reward selectively facilitated the speed of inhibitory control, but without significantly altering response execution measures, and did so similarly across groups. The data bear on a

number of issues, but particularly how the P-ADHD and S-ADHD groups differ and whether there are significant differences between children with TBI with and without identifiable S-ADHD.

Group Effects

A direct comparison of the ADHD groups allowed us to determine whether or not P-ADHD and S-ADHD share a similar cognitive-behavioral expression. Participants in the P-ADHD group exhibited poorer inhibitory control relative to typically developing children, but the S-ADHD group did not differ significantly from the Control group in terms of cancellation or restraint inhibition. However, their performance on the cancellation task was intermediate between the control, TBI, and P-ADHD groups. That is, they exhibited statistically similar inhibitory control performance relative to the Control group, but they also did not differ from the most impaired group, suggesting abnormal cancellation inhibitory control that is not as severe as the impairment exhibited by children with P-ADHD. Schachar, Levin and colleagues (2004) reported an inhibitory control deficit in children with S-ADHD and a severe TBI relative to controls. Because only one participant in our S-ADHD group had incurred a severe TBI, it remains unclear whether a group of children with more severe injuries would exhibit poorer inhibitory control compared to those with less severe injuries and/or those with P-ADHD.

Previous studies have shown that children with S-ADHD experience deficits in cancellation inhibition relative to typically developing children, but similar SSRT relative to children with only TBI (who were also impaired relative to controls; Konrad et al., 2000a, 2000b). The Konrad et al. (2000a, 2000b) studies included children who had incurred their injuries 6 months to 6 years prior to testing, thereby including participants in the acute phase of injury who might have experienced greater inhibition problems than those in the chronic phase of injury (Leblanc et al., 2005). Rapid functional improvement occurs soon after a TBI, especially in severe injuries, with more gradual recovery over the first few years post-TBI (Yeates, 2000). Performance in our TBI and S-ADHD groups may represent some recovery of inhibitory control abilities over time, which, to the extent that it occurred, would have attenuated group differences.

Contrary to expectations, the TBI group, who were all in the chronic phase of recovery, exhibited poor cancellation performance relative to Controls. Future studies are needed to examine whether injury location, area, and/or volume may account for these differences. Disrupted inhibition in P-ADHD may be related to abnormalities of frontostriatal circuitry (e.g. Aron & Poldrack, 2005; Kieling, Goncalves, Tannock, & Castellanos, 2008) including volume reductions and functional abnormalities in the basal ganglia (caudate nucleus and globus pallidus) and parts of the prefrontal cortex (e.g., Castellanos et al., 1996; Durston et al., 2004; Filipek et al., 1997; Rubia et al., 2010; Rubia et al., 1999; Seidman et al., 2006; Sowell et al., 2003; Vaidya et al., 2005). Children with TBI, with and without S-ADHD, who have lesions to the right ventrolateral prefrontal cortex and basal ganglia (especially the striatum) would be expected to exhibit longer SSRT on the cancellation task without significant deficits on the go trials (e.g., Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Chambers et al., 2007; Chevrier, Noseworthy, & Schachar, 2007; Rieger, Gauggel, & Burmeister, 2003). Conversely, participants with damage to the dorsal aspects of the prefrontal cortex, especially to the left side, may exhibit greater impairments with restraint (Levin et al., 1993).

Both ADHD groups were more variable than controls in their responses to go stimuli, implying that stability of response execution is disrupted to a similar degree in developmental and acquired disorders of attention. The inconsistent pattern of responding in the participants with P-ADHD is in line with a number of studies (e.g. Berwid et al., 2005;

Lijffijt, Kenemans, Verbaten, & Engeland, 2005; Schachar, Tannock, Marriott, & Logan, 1995), and may reflect problems in sustained attention and concentration, motor timing (Rubia, et al., 1999; Rubia, et al., 2001; Rubia et al., 2003) and response regulation (Rubia, Smith, & Taylor, 2007). Our study adds the new information that children with S-ADHD have unstable response execution. The exact mechanism of this problem in the TBI groups is unknown. Speed of response execution did not differ between the TBI and S-ADHD groups, so slower cognitive processing (Kalff et al., 2005) and slower motor speed (Van Meel, Oosterlaan, Heslenfeld, & Sergeant, 2005) can be excluded as possible mediators of group differences in response variability (SDRT).

Reward effects

Increases in reward improved the latency to cancel and restrain a response in all groups. This is consistent with our previous study that showed that increasing the reward magnitude for successful inhibition improved cancellation and restraint in typically developing children and adolescents (Sinopoli, Schachar, & Dennis, 2011). We extended that study by showing that reward facilitated inhibitory control in children with TBI (with and without S-ADHD) and those with P-ADHD, and showed that all groups benefited similarly from reward. That reward did not affect the speed of response execution confirms that the reward effects on SSRT are not merely a result of a general increase in the speed of information processing. How reward facilitates inhibitory control is not known. Reward may interact directly with the inhibitory control system, or act indirectly via a separate system that in turn influences the relationship between reward and inhibition substrates (Padmala & Pessoa, 2010). As elaborated elsewhere (Sinopoli et al., 2011), rewards may serve to increase arousal, attention, the hedonic value associated with a response, learning about stimuli-response associations, and/or the incentive salience of a stimulus associated with a response (see also Berridge, 2007).

Given that reward facilitated inhibition in children and adolescents with TBI, the mesolimbic dopamine system may be intact in the TBI and S-ADHD groups, or at least able to interact adaptively with the inhibition system. Our findings are in line with previous research that also showed the beneficial effects of rewards on inhibitory control performance in TBI and S-ADHD groups (Konrad et al., 2000b), and with a study that showed that increasing the magnitude of monetary incentives improved prospective memory in children with mild or severe TBI (McCauley, McDaniel, Pedroza, Chapman, & Levin, 2009). In neither the present study nor the Konrad et al. (2000b) or McCauley et al. (2009) studies is it clear whether rewards stimulated greater allocation of specific cognitive resources to increase proficiency, or whether reinforcement simply served to increase general arousal. The former is likely to be more correct. Relative to the Control group, the TBI and S-ADHD groups continued to exhibit greater variability in go responses in both stop signal tasks, regardless of reward conditions. Furthermore, MRT was unaffected by reward in both versions of the stop signal tasks, which indicates that reward did not generally increase arousal.

By selectively rewarding successful inhibition and including a neutral, non-reward condition, we avoided the methodological shortcomings of previous studies, and revealed that rewards facilitated inhibitory control in the P-ADHD group across tasks (see also Konrad et al., 2000b). These linear improvements in inhibitory control are consistent with previous findings showing that increasing the value of reinforcement improves SSRT (Slusarek, Velling, Bunk, & Eggers, 2001; Huang-Pollock et al., 2007), and also that children with P-ADHD make advantageous decisions with high magnitude (Luman, Oosterlaan, Knol, & Sergeant, 2008) or high frequency (Toplak, Jain, & Tannock, 2005) reinforcement. Children with P-ADHD may use immediate rewards to motivate, decrease distractibility, and focus attention (Sonuga-Barke, 2002). Here, high reward did not

normalize the stopping deficit exhibited by the P-ADHD group, so either the inhibitory control deficit in P-ADHD is too profound for rewards to significantly improve it, or children with ADHD, despite their preference for immediate incentive, have a higher than normal reward threshold (Douglas, 1999; Haenlein & Caul, 1987; Quay, 1997). Each hypothesis may account for the results, given both the lack of group interaction with reward and the improvements in SSRT with reward.

Limitations

The order of task administration affected the accuracy of go responses and the percentage of responses that were successfully inhibited, possibly reflecting participant fatigue or practice effects as the testing session progressed. Our main findings, namely the group and reward effects on SSRT, were not qualified by order effects, suggesting robustness in the results.

The S-ADHD group was identified on the basis of informant endorsement of clinically significant ADHD symptoms that met DSM-IV-TR criteria for the disorder. It is not clear, however, that individual psychiatric interviews would have improved the relation between diagnosis and inhibitory control outcome (see Willcutt et al., 2005).

Following the procedure used in previous studies (e.g., Konrad et al., 2000a, 2000b; Schachar, Levin, et al., 2004), we explored the presence of P-ADHD in the S-ADHD and TBI groups during the initial recruitment interview by probing for the presence of pre-injury DSM-IV-TR ADHD symptoms required for a diagnosis of P-ADHD. The number of participants in the S-ADHD group was much smaller than the sample sizes of the other participant groups. While a larger S-ADHD sample would have been welcome, our prevalence rate for S-ADHD is exactly that reported in the literature for chronic-phase TBI (18%; e.g., Gerring et al., 1998; Max et al., 2005b). In addressing the S-ADHD sample size, we considered a number of adjustments, including subtracting cases from cells with greater n until all cells were equal (Tabachnick & Fidell, 2007) to equate group sizes. We decided against this approach because artificially equalizing group sizes might have distorted group differences and limited generalizability (given that the unequal n in the current study represents actual population incidence of S-ADHD; Tabachnick & Fidell, 2007). Although greater inhibitory control deficits may have been predicted in children with S-ADHD and more severe injuries (see Schachar, Levin, et al., 2004), we could not explore this relation further.

Conclusions

Developmental and acquired forms of ADHD have distinct inhibitory control deficits. Children and adolescents with P-ADHD experienced greater difficulties with both cancellation and restraint than those with S-ADHD, which is congruent with the idea that poor inhibitory control is characteristic of children with a developmental form of P-ADHD (Crosbie & Schachar, 2001), so that a longer SSRT may be a candidate endophenotype only for this specific group of children (Crosbie et al., 2008). We did find, however, that the SSRTs of the S-ADHD group were not different from those of the P-ADHD group, which suggests that inhibition is not intact in children with S-ADHD and may represent a phenocopy of the behaviour exhibited by children with the developmental form of the disorder.

The data have some treatment implications. Stimulant medication ameliorates P-ADHD symptoms and improves the latency of cancellation (reviewed in Dennis, Sinopoli, Fletcher, & Schachar, 2008). Although stimulant effects on attention after childhood TBI are attenuated and time-limited, children with TBI do exhibit greater improvements in cognition relative to placebo (Jin & Schachar, 2004; Mahalick et al., 1998), so stimulants may have

some value in ameliorating the self-regulation difficulties in children with S-ADHD, and in stabilizing their inconsistent pattern of responses. Current behavioral therapies for P-ADHD utilize rewards and response costs to modify behaviour (Oosterlaan & Sergeant, 1998), and the focus of behavioral therapy should be on training children with ADHD to achieve (and maintain) an adequate motivational state to help reduce impulsivity, thus strengthening inhibitory control and other regulatory processes (see Luman, Oosterlaan, & Sergeant, 2005). Our data suggests that children and adolescents with TBI and S-ADHD would also benefit from similar behavioral interventions. Given the lack of group by reward interaction, however, it seems unlikely that incentives would fully normalize this deficit. Children in the Control group also benefited from rewards, with low levels of reward optimizing their inhibitory processes.

More generally, our study exemplifies the recent call (Levy & Ebstein, 2008) for principled and systematic comparisons of core cognitive phenotypes across disorders to inform cognitive processes, putative brain mechanisms, and treatment planning. S-ADHD as a result of childhood TBI results in inconsistent responding to go stimuli and inhibition performance that is atypical and intermediate between that of typically developing participants and a P-ADHD group. Unlike children with P-ADHD, children with S-ADHD do not show significant difficulties with restraint inhibition, similar to children with TBI without attention difficulties. Despite similarities in clinical manifestation, S-ADHD and P-ADHD appear to have different cognitive-behavioral phenotypes.

Acknowledgments

Funding was provided by the Ontario Neurotrauma Foundation to Katia J. Sinopoli (2007-ABI-PHD-565, The Regulation of Thoughts and Actions in Children Following Traumatic Brain Injury). This research was also supported by an NIH IRO1 grant to Maureen Dennis (HD04946, Social Outcomes in Pediatric Traumatic Brain Injury). We thank Shirley Chen for her help with the stop signal task, and Renee Farrell and Alexandra Basile for their help with scoring and coding of demographic and questionnaire information.

References

- American Psychiatric Association. The diagnostic and statistical manual of mental disorders, 4th edition text revision (DSM-IV-TR). Washington, DC: Author; 2000.
- Aron AR, Poldrack RA. The cognitive neuroscience of response inhibition: relevance for genetic research in attention-deficit/hyperactivity disorder. *Biological Psychiatry*. 2005; 57:1285–1292. [PubMed: 15950000]
- Aron AR, Fletcher PC, Bullmore ET, Sahakian BJ, Robbins TW. Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience*. 2003; 6:115–116.
- Band, GPH. Preparation, adjustment, and inhibition responses. (unpublished doctoral dissertation). Amsterdam: University of Amsterdam; 1997.
- Band GP, van der Molen MW, Logan GD. Horse-race model simulations of the stop-signal procedure. *Acta Psychologica*. 2003; 112:105–142. [PubMed: 12521663]
- Berridge KC. The debate over dopamine's role in reward: the case for incentive salience. *Psychopharmacology*. 2007; 191:391–431. [PubMed: 17072591]
- Berwid OG, Curco Kera EA, Marks DJ, Santra A, Bender HA, Halperin JM. Sustained attention and response inhibition in young children at risk for Attention-Deficit/Hyperactivity Disorder. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*. 2005; 46:1219–1229.
- Castellanos FX, Giedd JN, Marsh WL, Hamburger SD, Vaituzis AC, Dickstein DP, et al. Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry*. 1996; 53:607–616. [PubMed: 8660127]
- Catroppa C, Anderson V, Stargatt R. A prospective analysis of the recovery of attention following pediatric head injury. *Journal of the International Neuropsychological Society*. 1999; 5:48–57. [PubMed: 9989024]

- Chambers CD, Bellgrove MA, Gould IC, English T, Garavan H, McNaught EM, et al. Dissociable neural mechanisms of cognitive control in prefrontal and premotor cortex. *Journal of Neurophysiology*. 2007; 98:3638–3647. [PubMed: 17942624]
- Chevrier AD, Noseworthy MD, Schachar R. Dissociation of response inhibition and performance monitoring in the stop signal task using event-related fMRI. *Human Brain Mapping*. 2007; 28:1347–1358. [PubMed: 17274022]
- Cicerone KD. Attention deficits and dual task demands after mild traumatic brain injury. *Brain Injury*. 1996; 10:79–89. [PubMed: 8696318]
- Conners, CK. Conners 3rd edition. Toronto: Multi-Health Systems, Inc.; 2008.
- Crone EA, Jennings JR, van der Molen MW. Sensitivity to interference and response contingencies in attention-deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry*. 2003; 44:214–226. [PubMed: 12587858]
- Crosbie J, Perusse D, Barr CL, Schachar RJ. Validating psychiatric endophenotypes: Inhibitory control and attention deficit hyperactivity disorder. *Neuroscience and Biobehavioral Reviews*. 2008; 32:40–55. [PubMed: 17976721]
- Crosbie J, Schachar R. Deficient inhibition as a marker of familial ADHD. *American Journal of Psychiatry*. 2001; 158:1884–1890. [PubMed: 11691696]
- Dennis M, Sinopoli KJ, Fletcher JM, Schachar R. Puppets, robots, critics, and actors within a taxonomy of attention for developmental disorders. *Journal of the International Neuropsychological Society*. 2008; 14:673–690. [PubMed: 18764966]
- Dennis, M.; Wilkinson, M.; Koski, L.; Humphreys, R. Attention deficits in the long term after childhood head injury. In: Broman, S.; Michel, ME., editors. *Traumatic head injury in children*. New York: Oxford University Press; 1995. p. 165-187.
- Desman C, Petermann F, Hampel P. Deficit in response inhibition in children with attention-deficit/hyperactivity disorder (ADHD): Impact of motivation? *Child Neuropsychology*. 2008; 14:483–503. [PubMed: 18982507]
- Douglas, VI. Cognitive control process in Attention-Deficit/Hyperactivity Disorder. In: Quay, HC.; Hogan, AE., editors. *Handbook of disruptive behavior disorders*. New York: Plenum Press; 1999. p. 105-138.
- Durston S, Hulshoff Pol HE, Schnack HG, Buitelaar JK, Steenhuis MP, Minderaa RB, et al. Magnetic resonance imaging of boys with attention-deficit/hyperactivity disorder and their unaffected siblings. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2004; 43:332–340. [PubMed: 15076267]
- Filipek PA, Semrud-Clikeman M, Steingard RJ, Renshaw PF, Kennedy DN, Biederman J. Volumetric MRI analysis comparing subjects having attention-deficit hyperactivity disorder with normal controls. *Neurology*. 1997; 48:589–601. [PubMed: 9065532]
- Gerring J, Brady K, Chen A, Quinn C, Herskovits E, Bandeen-Roche K, et al. Neuroimaging variables related to development of Secondary Attention Deficit Hyperactivity Disorder after closed head injury in children and adolescents. *Brain Injury*. 2000; 14:205–218. [PubMed: 10759038]
- Gerring JP, Brady KD, Chen A, Vasa R, Grados M, Bandeen-Roche KJ, et al. Premorbid prevalence of ADHD and development of secondary ADHD after closed head injury. *Journal of the American Academy Child Adolescent Psychiatry*. 1998; 37:647–654.
- Gomez R. Underlying processes in the poor response inhibition of children with Attention-Deficit/Hyperactivity Disorder. *Journal of Attention Disorders*. 2003; 6:111–122. [PubMed: 12821876]
- Haenlein M, Caul WF. Attention deficit disorder with hyperactivity: a specific hypothesis of reward dysfunction. *Journal of the American Academy of Child and Adolescent Psychiatry*. 1987; 26:356–362. [PubMed: 3597291]
- Herskovits EH, Megalooikonomou V, Davatzikos C, Chen A, Bryan RN, Gerring JP. Is the spatial distribution of brain lesions associated with closed-head injury predictive of subsequent development of attention-deficit/hyperactivity disorder? Analysis with brain-image database. *Radiology*. 1999; 213:389–394. [PubMed: 10551217]
- Hollingshead, AB. Four factor index of social status. New Haven: Department of Sociology, Yale University; 1975.

- Huang-Pollock CL, Mikami AY, Pfiffner L, McBurnett K. ADHD subtype differences in motivational responsivity but not inhibitory control: Evidence from a reward-based variation of the stop signal paradigm. *Journal of Clinical Child and Adolescent Psychology*. 2007; 36:127–136. [PubMed: 17484686]
- Iaboni F, Douglas VI, Baker AG. Effects of reward and response costs on inhibition in ADHD children. *Journal of Abnormal Psychology*. 1995; 104:232–240. [PubMed: 7897047]
- Jin C, Schachar R. Methylphenidate treatment of attention-deficit/hyperactivity disorder secondary to traumatic brain injury: a critical appraisal of treatment studies. *CNS Spectrums*. 2004; 9:217–226. [PubMed: 14999162]
- Kalff AC, De Sonneville LM, Hurks PP, Hendriksen JG, Kroes M, Feron FJ, et al. Speed, speed variability, and accuracy of information processing in 5 to 6-year old children at risk for ADHD. *Journal of the International Neuropsychological Society*. 2005; 11:173–183. [PubMed: 15962705]
- Kieling C, Goncalves RR, Tannock R, Castellanos FX. Neurobiology of attention deficit hyperactivity disorder. *Child and Adolescent Psychiatry Clinics of North America*. 2008; 17:285–307.
- Konrad K, Gauggel S, Manz A, Scholl M. Inhibitory control in children with traumatic brain injury (TBI) and children with attention deficit/hyperactivity disorder (ADHD). *Brain Injury*. 2000a; 14:859–875. [PubMed: 11076133]
- Konrad K, Gauggel S, Manz A, Scholl M. Lack of inhibition: a motivational deficit in children with attention deficit/hyperactivity disorder and children with traumatic brain injury. *Child Neuropsychology*. 2000b; 6:286–296. [PubMed: 11992192]
- Leblanc N, Chen S, Swank PR, Ewing-Cobbs L, Barnes M, Dennis M, et al. Response inhibition after traumatic brain injury (TBI) in children: impairment and recovery. *Developmental Neuropsychology*. 2005; 28:829–848. [PubMed: 16266251]
- Levin HS, Culhane KA, Mendelsohn D, Lilly MA, Bruce D, Fletcher JM, et al. Cognition in relation to magnetic resonance imaging in head-injured children and adolescents. *Archives of Neurology*. 1993; 50:897–905. [PubMed: 8363443]
- Levin HS, Hanten G, Zhang L, Swank PR, Hunter J. Selective impairment of inhibition after TBI in children. *Journal of Clinical and Experimental Neuropsychology*. 2004; 26:589–597. [PubMed: 15370381]
- Levy Y, Ebstein RP. Research review: crossing syndrome boundaries in the search for brain endophenotypes. *Journal of Child Psychology and Psychiatry*. 2008; 50:657–668. [PubMed: 19175806]
- Lijffijt M, Kenemans JL, Verbaten MN, van Engeland H. A meta-analytic review of stopping performance in attention-deficit/hyperactivity disorder: deficient inhibitory motor control? *Journal of Abnormal Psychology*. 2005; 114:216–222. [PubMed: 15869352]
- Logan, GD. On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In: Dagenbach, D.; Carr, TH., editors. *Inhibitory processes in attention, memory, and language*. San Diego: Academic Press; 1994. p. 189–239.
- Logan GD, Cowan WB. On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review*. 1984; 91:295–327.
- Luman M, Oosterlaan J, Knol DL, Sergeant JA. Decision-making in ADHD: sensitive to frequency but blind to the magnitude of penalty. *Journal of Child Psychology and Psychiatry*. 2008; 49:712–722. [PubMed: 18422549]
- Luman M, Oosterlaan J, Sergeant JA. The impact of reinforcement contingencies on AD/HD: a review and theoretical appraisal. *Clinical Psychology Review*. 2005; 25:183–213. [PubMed: 15642646]
- Mahalick DM, Carmel PW, Greenberg JP, Molofsky W, Brown JA, Heary RG, et al. Psychopharmacologic treatment of acquired attention disorders in children with brain injury. *Pediatric Neurosurgery*. 1998; 29:121–126. [PubMed: 9838263]
- Max JE, Lindgren SD, Knutson C, Pearson CS, Ihrig D, Welborn A. Child and adolescent traumatic brain injury: correlates of disruptive behaviour disorders. *Brain Injury*. 1998; 12:41–52. [PubMed: 9483336]
- Max JE, Schachar RJ, Levin HS, Ewing-Cobbs L, Chapman SB, Dennis M, et al. Predictors of attention-deficit/hyperactivity disorder within 6 months after pediatric traumatic brain injury.

- Journal of the American Academy of Child and Adolescent Psychiatry. 2005a; 44:1032–1040. [PubMed: 16175108]
- Max JE, Schachar RJ, Levin HS, Ewing-Cobbs L, Chapman SB, Dennis M, et al. Predictors of secondary attention-deficit/hyperactivity disorder in children and adolescents 6 to 24 months after traumatic brain injury. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2005b; 44:1041–1049. [PubMed: 16175109]
- McCauley SR, McDaniel MA, Pedroza C, Chapman SB, Levin HS. Incentive effects on event-based prospective memory performance in children and adolescents with traumatic brain injury. *Neuropsychology*. 2009; 23:201–209. [PubMed: 19254093]
- Oosterlaan J, Sergeant JA. Effects of reward and response cost on response inhibition in AD/HD, disruptive, anxious, and normal children. *Journal of Abnormal Child Psychology*. 1998; 26:161–174. [PubMed: 9650623]
- Padmala S, Pessoa L. Interactions between cognition and motivation during response inhibition. *Neuropsychologia*. 2010; 48:558–565. [PubMed: 19879281]
- Quay HC. Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*. 1997; 25:7–13. [PubMed: 9093895]
- Rieger M, Gauggel S, Burmeister K. Inhibition of ongoing responses following frontal, nonfrontal, and basal ganglia lesions. *Neuropsychology*. 2003; 17:272–282. [PubMed: 12803433]
- Rubia K, Cubillo A, Smith AB, Woolley J, Heyman I, Brammer MJ. Disorder-specific dysfunction in right inferior prefrontal cortex during two inhibition tasks in boys with attention-deficit hyperactivity disorder compared to boys with obsessive-compulsive disorder. *Human Brain Mapping*. 2010; 31:287–299. [PubMed: 19777552]
- Rubia K, Overmeyer S, Taylor E, Brammer M, Williams SC, Simmons A, et al. Hypofrontality in attention deficit hyperactivity disorder during higher-order motor control: a study with functional MRI. *American Journal of Psychiatry*. 1999; 156:891–896. [PubMed: 10360128]
- Rubia K, Russell T, Overmeyer S, Brammer MJ, Bullmore ET, Sharma T, et al. Mapping motor inhibition: Conjunctive brain activations across different versions of Go/No-Go and Stop tasks. *Neuroimage*. 2001; 12:250–261. [PubMed: 11162266]
- Rubia K, Smith AB, Brammer MJ, Taylor E. Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *Neuroimage*. 2003; 30:351–358. [PubMed: 14527595]
- Rubia K, Smith A, Taylor E. Performance of children with attention deficit hyperactivity disorder (ADHD) on a test battery of impulsiveness. *Child Neuropsychology*. 2007; 13:276–304. [PubMed: 17453834]
- Schachar RJ, Chen S, Logan GD, Ornstein TJ, Crosbie J, Ickowicz A, et al. Evidence for an error monitoring deficit in attention deficit hyperactivity disorder. *J Abnormal Child Psychology*. 2004; 32:285–293.
- Schachar R, Levin HS, Max JE, Purvis K, Chen S. Attention deficit hyperactivity disorder symptoms and response inhibition after closed head injury in children: Do preinjury behavior and injury severity predict outcome? *Developmental Neuropsychology*. 2004; 25:179–198. [PubMed: 14984334]
- Schachar R, Logan GD, Robaey P, Chen S, Ickowicz A, Barr C. Restraint and cancellation: multiple inhibition deficits in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*. 2007; 35:229–238. [PubMed: 17351752]
- Schachar R, Tannock R, Marriott M, Logan G. Deficient inhibitory control in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*. 1995; 23:411–437. [PubMed: 7560554]
- Scheres A, Oosterlaan J, Sergeant JA. Response inhibition in children with DSM-IV subtypes of AD/HD and related disruptive disorders: the role of reward. *Child Neuropsychology*. 2001; 7:172–189. [PubMed: 12187474]
- Seidman LJ, Valera EM, Makris N, Monuteaux MC, Boriol DL, Kelkar K, et al. Dorsolateral prefrontal and anterior cingulate cortex volumetric abnormalities in adults with attention-deficit/hyperactivity disorder identified by magnetic resonance imaging. *Biological Psychiatry*. 2006; 60:1071–1080. [PubMed: 16876137]

- Shanahan MA, Pennington BF, Willcutt EW. Do motivational incentives reduce the inhibition deficit in ADHD? *Developmental Neuropsychology*. 2008; 33:137–159. [PubMed: 18443974]
- Sinopoli KJ, Schachar R, Dennis M. Reward improves cancellation and restraint inhibition across childhood and adolescence. *Developmental Psychology*. (in press).
- Slomine BS, Salorio CF, Grados MA, Vasa RA, Christensen JR, Gerring JP. Differences in attention, executive functioning, and memory in children with and without ADHD after severe traumatic brain injury. *Journal of the International Neuropsychological Society*. 2005; 11:645–653. [PubMed: 16212692]
- Slusarek M, Velling S, Bunk D, Eggers C. Motivational effects on inhibitory control in children with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2001; 40:355–363. [PubMed: 11288778]
- Sonuga-Barke EJ. Psychological heterogeneity in AD/HD—a dual pathway model of behaviour and cognition. *Behavioural Brain Research*. 2002; 130:29–36. [PubMed: 11864715]
- Sowell ER, Thompson PM, Holmes CJ, Jernigan TL, Toga AW. In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*. 1999; 2:859–861.
- Stevens J, Quittner AL, Zuckerman JB, Moore S. Behavioral inhibition, self-regulation of motivation, and working memory in children with attention deficit hyperactivity disorder. *Developmental Neuropsychology*. 2002; 21:117–139. [PubMed: 12139195]
- Tabachnick, BG.; Fidell, LS. *Using Multivariate Statistics*, 5th edition. Boston: Allyn and Bacon; 2007.
- Tamm L, Carlson CL. Task demands interact with the single and combined effects of medication and contingencies on children with ADHD. *Journal of Attention Disorders*. 2007; 10:372–380. [PubMed: 17449836]
- Teasdale G, Jennet B. Assessment of coma and impaired consciousness. A practical scale. *Lancet*. 1974; 2:81–84. [PubMed: 4136544]
- Toplak ME, Jain U, Tannock R. Executive and motivational processes in adolescents with Attention-Deficit-Hyperactivity Disorder (ADHD). *Behavioral Brain Function*. 2005; 1:8.
- Vaidya CJ, Bunge SA, Dudukovic NM, Zalecki CA, Elliott GR, Gabrieli JD. Altered neural substrates of cognitive control in childhood ADHD: evidence from functional magnetic resonance imaging. *American Journal of Psychiatry*. 2005; 162:1605–1613. [PubMed: 16135618]
- Van Meel CS, Oosterlaan J, Heslenfeld DJ, Sergeant JA. Motivational effects on motor timing in attention-deficit/hyperactivity disorder. *American Academy of Child and Adolescent Psychiatry*. 2005; 44:451–460.
- Verbruggen F, Logan GD. Response inhibition in the stop-signal paradigm. *Trends in Cognitive Sciences*. 2008a; 12:418–424. [PubMed: 18799345]
- Verbruggen F, Logan GD. Models of response inhibition in the stop-signal and stop-change paradigms. *Neuroscience and Biobehavioral Reviews*. 2008b; 33:647–661. [PubMed: 18822313]
- Wassenberg R, Max JE, Lindgren SD, Schatz A. Sustained attention in children and adolescents after traumatic brain injury: relation to severity of injury, adaptive functioning, ADHD and social background. *Brain Injury*. 2004; 18:751–764. [PubMed: 15204316]
- Willcutt EG, Doyle AE, Nigg JT, Faraone SV, Pennington BF. Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review. *Biological Psychiatry*. 2005; 57:1336–1346. [PubMed: 15950006]
- Williams BR, Ponsesse JS, Schachar RJ, Logan GD, Tannock R. Development of inhibitory control across the life span. *Developmental Psychology*. 1999; 35:205–213. [PubMed: 9923475]
- Wodka EL, Mahone EM, Blankner JG, Gidley Larson JC, Fotadar S, Denckla MB, et al. Evidence that response inhibition is a primary deficit in ADHD. *Journal of Clinical and Experimental Neuropsychology*. 2007; 29:345–356. [PubMed: 17497558]
- Yeates KO, Armstrong K, Janusz J, Taylor HG, Wade S, Stancin T, Drotar D. Long-term attention problems in children with traumatic brain injury. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2005; 44:574–584. [PubMed: 15908840]
- Yeates, KO. Closed-head injury. In: Yeates, KO.; Taylor, HG.; Ris, MD., editors. *Pediatric Neuropsychology: Research, Theory, and Practice*. New York: The Guilford Press; 2000. p. 92-105.

Young SE, Friedman NP, Miyake A, Willcutt EG, Corley RP, Haberstick BC, Hewitt JK. Behavioral disinhibition: liability of externalizing spectrum disorders and its genetic and environmental relation to response inhibition across adolescence. *Journal of Abnormal Psychology*. 2009; 118(1): 117–130. [PubMed: 19222319]

Figure 1.

Cancellation and Restraint models of the stop signal task (with and without rewards). Trials 1 and 2 represent the primary task in both versions, where a go signal (X in Trial 1, O in Trial 2) is presented following a fixation point. Participants were instructed to respond to these stimuli as quickly as possible by making the appropriate button press on a handheld controller. An inhibitory control trial is depicted in Trial 3. In the cancellation version (left panel), the initial auditory stop signal was presented 250 milliseconds (ms) following onset of the go signal. If a participant was able to successfully inhibit his/her response, then the delay was increased by 50 ms on the subsequent stop trial to increase the level of difficulty, but if the participant was unable to inhibit his/her response, then the delay was decreased by 50 ms on the subsequent stop trial to make cancellation easier. In the restraint version (right panel), the stop signal was always presented at the same time as the go signal (the delay between the onset of the go and stop signal was zero). In both versions of the task, the feedback following the inhibition trials were identical: failed inhibition (i.e., going instead of stopping or withholding) resulted in no feedback in the NO condition, and in a failure to win points in the LOW and HIGH conditions; successful inhibition in the NO condition also resulted in no feedback, but each successful inhibition in the LOW and HIGH conditions resulted in the receipt of 2 or 10 points, respectively.

Table 1

Participant Characteristics

GROUP	AGE AT TEST (years)	AGE AT INJURY (years)	TIME SINCE INJURY (years)	WASI IQ SCORE	SES
CONTROL	12.35 (3.4)	--	--	112.3 (11.0) *	52.8 (6.2) **
TBI	11.91 (3.4)	8.3 (3.6)	3.7 (1.5)	105.0 (10.5)	44.1 (10.1)
MILD	11.9 (2.9)	8.1 (2.9)	3.7 (1.4)	108.3 (9.6)	44.4 (11.1)
MODERATE	12.2 (4.3)	7.7 (5.1)	4.5 (1.1)	98.0 (10.3)	39.4 (11.4)
SEVERE	11.9 (3.4)	8.7 (4.3)	3.4 (1.7)	102.47 (10.8)	45.0 (8.5)
S-ADHD	11.8 (2.5)	7.2 (2.4)	4.6 (1.2)	99.0 (14.3)	44.6 (10.5)
MILD	11.1 (2.6)	6.5 (2.5)	4.5 (1.3)	102.2 (12.3)	43.7 (11.2)
MODERATE	13.2 (2.3)	8.9 (2.6)	4.3 (0.4)	101.0 (17.0)	47.0 (15.5)
SEVERE	13.9	8.1	5.8	76.0	45.0
P-ADHD	10.9 (3.2)	--	--	108.3 (17.0)	46.2 (11.5)

All scores represented as means (standard deviation)

* Control group vs. TBI ($p=0.008$) and S-ADHD ($p=0.004$) groups

** Control group vs. TBI ($p<0.001$), S-ADHD ($p=0.016$), and P-ADHD ($p=0.014$) groups

Table 2**Inhibitory Control Measures on the Cancellation and Restraint Tasks**

Dependent Variable	Group			
	Control	TBI	S-ADHD	P-ADHD
SSRT-C				
NO *	216.92 (18.9) [^]	250.35 (17.9)	270.58 (42.9)	304.66 (26.7)
LOW *	191.16 (13.4) [^]	219.35 (12.7)	232.17 (30.5)	276.74 (19.0)
HIGH *	191.28 (13.0) [^]	214.58 (12.3)	174.25 (29.5)	232.99 (18.3)
PI-C				
NO	56.63 (1.2)	53.38 (1.2)	50.92 (2.8)	54.91 (1.8)
LOW	52.35 (0.9)	53.20 (0.8)	52.89 (1.9)	53.40 (1.2)
HIGH	52.72 (0.9)	50.93 (0.8)	55.57 (2.0)	48.45 (1.2)
SSRT-R				
NO **	379.79 (16.1) [^]	425.60 (15.3)	430.85 (36.6)	421.72 (22.8)
LOW **	367.43 (12.5) [^]	382.14 (11.9)	390.25 (28.5)	392.55 (17.7)
HIGH	352.32 (13.7) [^]	398.89 (13.0)	361.12 (31.2)	393.81 (19.4)
PI-R				
NO	88.71 (2.9)	84.30 (2.7)	81.75 (6.6)	82.02 (4.1)
LOW	94.01 (2.3)	88.93 (2.2)	89.95 (5.3)	86.85 (3.3)
HIGH	93.77 (1.6)	90.49 (1.5)	92.50 (3.6)	88.00 (2.2)

All scores represented as means (standard deviation)

Legend: SSRT-C = stop signal reaction time in milliseconds, cancellation task; PI-C = percent inhibition, cancellation task; SSRT-R = stop signal reaction time in milliseconds, restraint task; PI-R = percent inhibition, restraint task; NO = neutral reward condition; LOW = low reward condition; HIGH = high reward condition

* SSRT-C decreased linearly from the NO to LOW to HIGH conditions

** SSRT-R was faster in the reward conditions (LOW and HIGH) relative to the NO condition

[^] The Control group vs. TBI and P-ADHD groups

Table 3

Response Execution Measures on the Cancellation and Restraint Tasks

Dependent Variable	Group			
	Control	TBI	S-ADHD	P-ADHD
MRT-C				
NO	535.88 (15.3)	577.11 (14.4)	524.56 (34.6)	554.40 (21.6)
LOW	547.38 (15.3)	568.98 (14.5)	529.84 (37.8)	548.24 (21.6)
HIGH	547.12 (16.2)	562.00 (15.3)	546.22 (36.8)	551.20(22.9)
SDRT-C				
NO	118.21 (8.1)*	148.14 (7.7)	139.70 (18.4)	158.30 (11.5)
LOW	124.82 (6.7)*	142.87 (6.3)	153.09 (15.1)	135.74 (9.4)
HIGH	124.67 (6.2)*	140.55 (5.9)	137.56 (14.1)	151.41 (8.8)
PCR-C				
NO	96.88 (0.6)	97.63 (0.6)	95.48 (1.4)	93.20 (0.9)**
LOW	96.97 (0.9)	96.26 (0.8)	92.91 (2.0)	90.15 (1.3)**
HIGH	96.96 (0.9)	95.23 (0.9)	94.38 (2.1)	91.09 (1.3)**
MRT-R				
NO	502.52 (13.9)	542.82 (13.1)	563.09 (31.5)	540.06 (19.6)
LOW	492.68 (14.2)	522.14 (13.4)	522.23 (32.3)	527.10 (20.1)
HIGH	488.16 (17.7)	533.03 (13.9)	537.50 (33.5)	535.23 (20.8)
SDRT-R				
NO	118.21 (8.1)*	134.01 (8.8)	168.57 (21.0)	148.61 (13.1)
LOW	112.89 (8.0)*	123.56 (7.5)	139.15 (18.1)	148.48 (11.2)
HIGH	112.22 (8.3)*	127.42 (7.9)	153.34 (18.9)	137.49 (11.7)
PCR-R				
NO	96.81 (1.0)	95.86 (0.9)	94.63 (2.2)	92.52 (1.4)**
LOW	95.36 (1.1)	95.19 (1.1)	95.83 (2.5)	91.27 (1.6)**
HIGH	95.61 (1.1)	93.46 (1.1)	91.29 (2.5)	91.82 (1.6)**

All scores represented as means (standard deviation)

Legend: MRT-C = mean (go) reaction time in milliseconds, cancellation task; SDRT-C = standard deviation of go reaction time in milliseconds, cancellation task; PCR-C = percent correct on the go task, cancellation task; MRT-R = mean (go) reaction time in milliseconds, restraint task; SDRT-R = standard deviation of go reaction time in milliseconds, restraint task; PCR-R = percent correct on the go task, restraint task; NO = neutral reward condition; LOW = low reward condition; HIGH = high reward condition

* Control group vs. all groups

** P-ADHD group vs. Control and TBI groups

Table 4

Linear Regression Results

Variable	Significant Predictor	B	Standard error of B	Adjusted R ²
MRT-C				
NO	Age at injury	−24.21	4.4	0.389
	Time since injury	−30.85	9.7	
LOW	Age at injury	−19.85	4.3	0.317
	Time since injury	−25.02	9.4	
HIGH	Age of injury	−8.98	1.8	0.336
SDRT-C				
NO	Age at injury	−9.37	2.5	0.201
LOW	Age at injury	−9.02	2.1	0.293
HIGH	Age at injury	−18.59	4.9	0.213
SSRT-C				
HIGH	Age at injury	−7.09	3.01	0.145
	GCS score	−4.75	2.2	
MRT-R				
NO	Age at injury	−22.19	3.8	0.414
	Time since injury	−22.18	8.3	
LOW	Age at injury	−23.54	3.7	0.473
	Time since injury	−20.84	8.3	
	GCS score	−5.9	2.8	
HIGH	Age of injury	−21.26	4.2	0.345
	Time since injury	−18.63	9.2	
SDRT-R				
NO	Age at injury	−10.03	2.9	0.148
LOW	Age at injury	−11.91	2.1	0.400
	GCS score	−3.21	1.6	
HIGH	Age at injury	−10.14	2.3	0.250
PCR-R				
HIGH	Age at injury	1.25	0.4	0.184
	Time since injury	1.67	0.8	
SSRT-R				
LOW	Age at injury	−14.77	3.6	0.223
HIGH	Age at injury	−15	4.1	0.186

Legend: MRT-C = mean (go) reaction time, cancellation task; SDRT-C = standard deviation of go reaction time, cancellation task; SSRT-C = stop signal reaction time, cancellation task; MRT-R = mean (go) reaction time in milliseconds, restraint task; SDRT-R = standard deviation of go reaction time in milliseconds, restraint task; PCR-R = percent correct on the go task, restraint task; SSRT-R = stop signal reaction time, restraint task; NO = neutral reward condition; LOW = low reward condition; HIGH = high reward condition