Gupta, R. & Kar, B. R. (2009). Development of attentional processes in normal and ADHD children. *Progress in Brain Research, 176,* 259-276.

Running Head: Development of Attention

Development of Attentional Processes in ADHD and Normal Children

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Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is a developmental disorder. Typical development of attentional processes is rapid during early childhood. ADHD results in impairment in response inhibition, error monitoring, attentional disengagement, executive attention, and delay aversion and may effect the ongoing development of these processes during childhood. We examined the development of attentional processes in children with ADHD and normal children. 240 Children (120 in each group) in the age rang of 6-9 years participated in the study. Four tasks: Stop-Signal, attentional disengagement, attention network, and choice delay task were administered. Stop signal reaction time, switch costs, conflict effect, and percentage choice of short delay reward was higher in ADHD group compared to normal group. Post error of slowing was less in ADHD children. Endogenous orienting effect was more in normal children compared to ADHD children. Different developmental trajectories were observed for control functions in normal children. Major development in response inhibition occurred in 7-8 years, error monitoring in 6-9 years, and attentional disengagement in 7-9 years. Late development in alerting network was observed in normal children at age 9 years. No developmental changes occurred on these control functions in ADHD children aged 6-9 years. Age related changes were observed on delay aversion between 6-9 years in normal children, while it changed between 6-7 years in ADHD children. Performance was not changed on orienting and conflict attentional networks in both the children except conflict effect reduced between 7-9 years in ADHD children under double cue condition. Conflict network was interacted with the alerting and orienting network in normal children, specifically conflict network interacted with the orienting network in younger children

(age 6 years) and with alerting network in older children (age 9 years). In ADHD group interaction between alerting and conflict network was observed only in the double cue condition. Together these results indicated that the deficits in control processes accumulate with age in ADHD children Present study favors the conceptual view of ADHD as a stable deficit in cognitive control functions, which are implicated in the pathology of ADHD. These results have theoretical implication for the theories of executive control and ADHD.

Key Words: ADHD, development, response inhibition, error monitoring, attentional disengagement, attentional networks, delay aversion, stable deficit

Introduction

Age related changes have been identified with control processes such as response inhibition, working memory, task switching, and error monitoring that are critical for perception and action. These processes also have been found to be deficient in certain developmental disorders such as Attention Deficit Hyperactivity Disorder (ADHD).

Development of Attention-Executive Processes in Normal Children

A number of the studies have examined the developmental trajectories of attention-executive processes in normal children (Bunge et al., 2002; Gupta et al., submitted, a). For example, it has been reported that inhibitory control develops over childhood and does not reach full maturity until 12 years of age or later (Bunge et al., 2002). Substantial improvement in inhibitory control occurs during childhood and it declines during late adulthood (Williams et al., 1999), suggesting an inverted-U-shaped relationship between inhibitory control and age. Paradigms such as Go-No-Go task, Stop-Signal task, and Stroop task have been used to study inhibitory control. Performance on Stroop-like tasks improves through 3-7 years of age (Gerstadt et al., 1994) and declines during late adulthood (Spieler et al., 1996). Christ et al. (2001) investigated the ability to inhibit a prepotent response and generate an incompatible response in individuals ranging from 6-82 years of age. They found that inhibitory control effect was larger in children and older adults than for young adults, and larger for older adults than children. They further argue that childhood is a critical period in terms of frontal lobe and cognitive development. Thus, changes in inhibitory control could have occurred within the age range of 6-15 year old age group. They divided the child group into two age groups: 6-9 and 10-15 years of age. Raw reaction time data suggested that younger children respond more slowly than

older children. To determine if the discrepancy in the magnitude of the effect was attributable to differences in processing speed rather than inhibitory control, the data were reanalyzed following proportional and z-score transformation. Results of the proportional score supported the raw reaction time data. However, using the more rigorous z-score procedure, group effect as well as interaction between group and condition (congruent vs incongruent) was not found to be significant. These findings indicated that early-age related difference in inhibitory control were due to difference in processing speed rather than true differences in inhibitory control.

It has been argued that inhibition of task set is one of the contributors to attentional disengagement measured by switch costs (Monsell, 2003). Cepeda et al. (2001) examined age related differences in task switching with respect to the processes responsible for preparation and interference control that underlie the ability to flexibly alternate between two different tasks. They observed larger switch costs among young children (7-9 years) and it decreased with age. Crone et al. (2006) found greater switch costs with young children (7-8 years of age) compared to adults for task switching with repeating responses. This age related difference decreased with an increase in the interval between the previous response and the upcoming stimulus. Young children experienced more interference from the previous stimulus-response (S–R) association, suggesting larger carry-over effects from the previous trial.

Errors may occur while switching between actions. Monitoring for such errors online and making subsequent adjustments in processing speed is important for cognitive control. Error monitoring is evident in the slowing of responses following errors (Post error slowing, PES) (Rabbit, 1966), which varies with age. PES varied with age within

the range of 7-16 years, older children slowed less than younger ones (Schachar et al., 2004). Kramer et al. (1994) found that elderly participants showed larger PES than younger adults following trials where an error had been made (50 ms vs. 21 ms). Together these studies indicate a curvilinear pattern of development in PES is indicated across the life span, showing a decrease as children get older followed by an increase among older adults. Posner and Rothbart (1998) found that children were able to detect an error as early as 48 months of age. Error monitoring was studied with event related potentials between 7-25 years of age and it has been found that error related negativity (ERN) (reflects unconscious detection of an error) amplitude increased with age. However, the error-positivity (*Pe*) amplitude (reflects conscious error recognition and performance adjustment after an error) did not change with age (Davies et al., 2004).

In addition to the specific higher order executive processes, development of attentional networks (alerting, orienting, and executive) from 4 years of age to adulthood has been reported (Rueda, et al., 2004a; 2004b). These studies indicate that there was a steady decline in overall reaction time from 4 years of age to adulthood. Improvement in conflict resolution was found until age seven. Alerting scores showed some improvement in late childhood and continued development between 10 year olds and adults. The orienting score was similar to adult levels at the youngest ages.

Attention-Executive Deficits in ADHD children

Attention-executive dysfunction characterized by deficits in response inhibition, error monitoring, attentional disengagement, attentional networks, and motivational style (delay aversion) in children with ADHD (see Pennington & Oznoff, 1996; Gupta et al., 2006, for reviews). Barkley (1997) suggested that response inhibition is the primary

deficit in ADHD, which in turn affects the other executive functions. The evidence supporting a deficiency in behavioral inhibition in ADHD comes from studies that used motor inhibition tasks, such as go-no-go task (Iaboni et al., 1995), the stop-signal task (Oosterlaan & Seregant, 1998), and delayed response tasks (Sonuga-Barke et al., 1992). Further support for deficient inhibitory control in ADHD is suggested by neuroimaging research indicating both structural and functional deficits in the right inferior frontal cortex (Aron & Poldrack, 2005). Schachar et al. (2004) studied error monitoring by looking at the slowing of responses after inhibition error (PES) in a Stop-Signal task. ADHD children slowed to a lesser extent after fewer inhibition failures suggesting deficits in error detection as well as in behavioral adjustment to errors. Cepeda et al. (2000) suggested that ADHD children show deficient control processes necessary for disengagement from one task and preparation for a subsequent task.

ADHD children also show impairments in an executive and alerting network due to the inability to maintain the alert state when no warning signal was used (Blane & Marrocco, 2004). In addition to executive function deficits children with ADHD are also characterized by a specific motivational style called delay aversion, which is the motivation to avoid delay and results in preference for small immediate over large delayed rewards (Sonuga-Barke, 2002).

Most of the studies have examined the development of attentional processes in normal children. Very few studies have investigated the development of attentional processes in ADHD children. To our knowledge only one study has examined the development of attentional processes particularly selective attention in ADHD children compared to normal children aged between 6-11 years (Brodeur & Pond, 2001). In this

study, two age groups of children (6-8 years and 9-11 years) with ADHD and normal children were tested using a timed computer task. The task consisted of identifying visual target stimuli under various distracter conditions. Children with ADHD were less efficient on the selective attention task than were children without ADHD, and older children were more efficient than younger children in both groups. Children without ADHD were influenced more by the nature of distracters than were children with ADHD. This study talked about only one component of attention such as selective attention. Yet another study, longitudinally examined the brain development especially the cortical maturation of ADHD children from 10 to 17 years of age. They estimated the cortical thickness at various cerebral points. They found that sequence of brain maturation in children with ADHD follows the normal pattern but is delayed by 2-3 years in prefrontal regions that are important for control of cognitive processes including attention and motor planning (Shaw et al., 2007). This study reported delay in the structural development of the brain in ADHD. There is no evidence on delay in functional maturation of the brain and cognitive processes in ADHD. In other words, it is still an open question whether ADHD is characterized by a delay in normal ongoing development of, or a stable deficit in control processes (Brocki & Bohlin, 2006). Brocki and Bohlin (2006) support the conceptual view of ADHD as a developmental delay, as ADHD symptoms changed with maturation. However, developmental delay in control processes in ADHD was not studied. In addition, they only included the non-clinical sample in their study. The key to understanding ADHD as either a developmental or a categorical disorder lies in comparing developmental trends in a normal group with a clinical ADHD group. Hence, a study on development of attentional processes in children

with a clinical diagnosis of ADHD was needed to understand if attentional processes in ADHD represent a delay or a complete deviation from typical development. In the present study we focused on the development of various attentional control functions such as response inhibition, attentional disengagement, error monitoring, attentional networks, and motivational style in ADHD compared to normal children aged 6-9 years. These control functions are important in developing a theory about ADHD (see Gupta et al., 2006 for review) and was found to be sensitive in the diagnosis of ADHD from normals and also helpful in differential diagnosis of ADHD with other developmental disorders such as Oppositional Defiant Disorder (ODD) (Gupta et al., submitted, b).

Previous studies have examined the development of control processes by employing a relatively small numbers of different of age groups among normal children (Johnstone et al., 2005; Cepeda et al., 2001). For example, Johnstone et al. (2005) examined 7-47 years old participants to study the development of response inhibition. Studies on task switching and error monitoring have examined fewer cases or coarse groupings of ages. For example, Cepeda et al. (2001) studied task switching with two groups: 7-9 and 10-12 year old children. Combining children with different ages makes it difficult to track developmental changes in these executive processes. We have examined four age levels (6-9 years) to closely evaluate developmental patterns of control processes among children with and without ADHD. Six to nine years of age is the period of school age, hence, most of the ADHD cases are reported in this age range. DSM-IV also suggests that ADHD should be diagnosed before the age of seven years. Examination of the different control functions in the same group of children enabled us to control for difference in experimental procedures used in different studies and participant

demographics. It is also important to determine whether development of control functions occurs in parallel in a particular age group. If so, then a possible interaction among these control functions may be implicated in the pathology underlying ADHD.

Method

Participants

120 children with ADHD (N = 120; 117 boys) and 120 normal children (N = 120; 114 boys) in the age range of 6-9 years (30 children in each age level) participated in the study. Participants were recruited from two districts of the state of Uttar Pradesh like Allahabad and Lucknow. The participants with ADHD were referred by consultant psychiatrists. Both the groups were matched on socioeconomic status. Practicing psychiatrists referred children with ADHD. All the participants with ADHD fulfilled the DSM-IV (APA, 1994) criteria for combined type ADHD. They scored the clinical cut-off (T>60) on Conners Parents Rating Scale-Revised Long form (CPRS-R:L, Conners, 2002). CPRS-R:L was also used to rule out any behavioral and attentional problems in normal children. 20% participants with ADHD had a co-morbid diagnosis of ODD. Numbers of children with co-morbid ODD were equally distributed across the different age groups and this subgroup did not differ in performance profile from the pure ADHD group. Therefore, data from children with pure ADHD as well as those with ADHD with comorbid ODD were analyzed together as a one group. All the children were average or above average in intellectual functions with scores in the range of 50-95 percentile on Colored Progressive Matrices (CPM) (Raven et al., 1998). Table 1 shows the demographic details of two groups of participants.

Procedure

Participants were tested individually on four cognitive tests. Order of the tests was randomized across participants. Each participant was seated in front of the computer at a distance of 60.0 cm and received instructions for each test. CPRS-R:L was administered to the parents. Whole assessment took approximately one hour per child. All the participants had normal or corrected to normal vision. A written informed consent was taken from the parents. The ethics committee of the Centre of Behavioral and Cognitive Sciences, University of Allahabad had approved the study.

Measures of Cognitive Functions

Attentional Network Test: Attention Networks

The child version of Attentional Network Test (ANT) (Rueda et al., 2004) was employed to examine the attentional networks: alerting, orienting, and executive networks. The target array was a yellow colored line drawing of either a single yellow fish or a horizontal row of five yellow fish, presented above or below fixation, over a blue–green background. The participant had to respond whether the central fish was pointing to the left or right by pressing the corresponding left or right key on the key board. The ANT consisted of a total of 24 practice trials and three experimental blocks of 48 trials each. Each trial represented one of 12 conditions in equal proportions: three target types (congruent, incongruent and neutral) and four cues (no cue, central cue, double cue and spatial cue) (Rueda et al., 2004). ANT test does not have invalid trials, hence, all the cues are endogenous cues. However, unlike the spatial cue, the double cue did not predict the target exactly because double cues appeared both the above and below the fixation and target appeared only on one location either above or below the fixation.

Hence, we argue that double cue may function as an exogenous cue. We expected a difference in performance in normal and ADHD children with respect to cue conditions, specifically with double and spatial cue. Therefore, in addition to orienting effect score (difference between center cue and spatial cue) of ANT, we also computed another orienting score by subtracting the median RT for double cue from the RT for central cue. Former orienting score was called as endogenous orienting and later as exogenous orienting. Alerting effect, orienting effect and conflict effect was calculated to measure alerting network, orienting network, and conflict network respectively. To find out the orienting and alerting scores per subject we computed the median RT per cue condition (across the flanker conditions). The alerting score was obtained by subtracting the median RT for the double cue from median RT for the no cue condition; the endogenous orienting score by subtracting the median RT for spatial cue from the RT for central cue; exogenous orienting score by subtracting the median RT for double cue from the RT for central cue. To obtain the conflict score we computed the participant's median RT for each flanker condition (congruent vs incongruent) (across cue conditions) and subtracted the congruent from the incongruent RTs. The mean score, across subjects, was then computed for each network.

Stop-Signal Test: Response Inhibition

The Stop-Signal Test (SST) involved two concurrent tasks. The primary or go task involved discrimination between X or an O presented in the centre of a computer screen for 1000 ms following a 500 ms fixation point. The go stimulus was followed by a blank screen for 2000 ms allowing 3000 ms for key press and total trial duration of 3500 ms. The secondary or stop task involved the presentation of a green visual circle indicating

that participants should not respond to the primary task. The Green circle occurred randomly and with equal frequency across blocks on 25% of trials. The session consisted of four blocks of 40 trials. We used a dynamic tracking procedure to set the timing of the circle (stop signal delay) (Logan, 1994). At the beginning of the task, then stop delay was set at 250 ms. If a participant was able to stop successfully, the delay was lengthened (by 50 ms) on the succeeding trial (see Schachar et al., 2004, for more details). Stop Signal Reaction Time (SSRT) and Post Error Slowing (PES) was calculated to measure response inhibition and error monitoring respectively.

Attentional Disengagement Test: Attentional Disengagement

Stimuli were presented at fixation. The four possible stimuli were either a single digit (1 or 3) or three digits (1 1 1 or 3 3 3). In other words, either one or three numeric 1s or 3s were presented. On each trial either the cue, "What number?" or the cue "How many?" appeared above the target stimulus. Participants were required to switch their attention between two different tasks with respect to the cue that appeared above the target stimulus: discriminating the value of a number presented on a computer screen or deciding how many numbers were present on the screen. Stimuli stayed on the screen until response was made. Feedback (100 Hz tone) was given whenever participants made an error. A practice session with 75 trials preceded the experimental session consisting of 200 trials. Switch cost (SC) was calculated to measure attentional disengagement.

Choice Delay Test: Delay Aversion

In the Choice Delay Test (CDT) participants were presented with a series of trials and asked to choose between a small reward (1 point) to be delivered after a short delay (1 second) or a large reward (2 points) to be delivered after a long delay (20 seconds). The

child could choose between a small, immediate reward and a large, delayed reward, and the total length of the trial depended on the percentage of choices for the large, delayed reward. A practice session with 5 trials proceeded the experimental session consisting of 30 trials. Percentage choice of long delay reward (%LDR) and short delay reward (%SDR) was calculated to measure delay aversion.

Data Analysis

For each score data were submitted to 2 (Group: Normal and ADHD) × 4 (age levels: 6, 7, 8, 9) between factor design.

Results

Age Effects

Response Inhibition: *Stop-Signal Reaction Time*

The analysis yielded a significant effect of group, $F(1, 232) = 307.1$, $p < .001$, with higher SSRT for ADHD (M = 646.0 ms) as compared to normals (M = 310.8 ms). The Effect of age was also significant, $F(3, 232) = 4.64$, $p < .01$. SSRT was more for 6 years old children and it decreased as age increased. To ensure whether the age effect was significant in both the groups, we performed one-way ANOVA with age as a between factor for both the groups separately. Age effect was found only in the normal group, $F(3, 116) = 8.05$, $p < .0001$. Tukey's HSD for post-hoc comparisons was performed. Performance improved in children between 7 and 8 years old, $F(1, 116) = 4.50$, $p < .01$.

Insert Figure 1 about here

Error Monitoring: *Post-Error Slowing*

The analysis yielded a significant effect of group, $F(1, 232) = 174.7$, $p < .001$, with lesser extent of slowing for ADHD children (M = 19.7 ms) compared to normals (M = 119.7 ms). The Effect of age was also significant, $F(3, 232) = 4.18$, $p < .01$, with children of 6 years being significantly slower. Performance speeded with age. Performance of 6 years old children was significantly different from the performance of 8 years old children, $F(1, 232) = 3.97$, $p < .05$. Group × age interaction effect was found significant, $F(3, 232) = 4.99$, $p < .01$. Post hoc comparisons indicated that performance of normal children changed from 6 to 8 years of age, $F(1, 232) = 6.07$, $p < .001$. There was no change in performance of ADHD children between 6 to 9 years of age.

Insert Figure 2 about here

Aattentional Disengagement: *Switch Costs*

Group effect was significant, $F(1, 232) = 391$, $p < .001$. SC was higher for ADHD (M = 633.4 ms) as compared to normals (M = 224.0 ms). Age effect was also significant, $F(3, 232) = 11.8$, $p < .01$. SC was more for 6 years old children and it decreased with increase in age. To ensure the age effect in both the groups, we performed one-way ANOVA with age as a between factor for both the groups separately. Age effect was found only in the normal group, $F(3, 116) = 21.0$, $p < .001$. Task switching improved from 7 to 8 years, $F(1, 116) = 4.15$, $p < .05$, and 8 to 9 years of age, $F(1, 116) = 4.37$, $p < .01$, in normal children.

Insert Figure 3 about here

Attentional Networks: *Alerting Effect, Endogenous and Exogenous Orienting Effect, Conflict Effect*

A Two-way ANOVA was computed with group and age as between factors in order to assay the developmental trend of each attentional network. Group effect was significant for endogenous orienting, $F(1, 232) = 9.44$, $p < .01$, and conflict score, $F(1, 232) = 8.36$, $p < .01$. Orienting effect was more for normal (M = 43.15 ms) compared to ADHD children (M = 19.91 ms). In addition, conflict effect was more for ADHD (M = 100.8 ms) compared to normal children (M = 73.2 ms). Age effect was not significant for any of the networks. Group × age interaction was significant only for alerting network, $F(3, 232) = 3.27$, $p < .05$. Post-hoc comparison indicated that performance of 9 year old normal children was marginally different from the performance of 9 year old ADHD children, $F(1, 232) = 4.13$, $p = 0.07$.

Insert Figure 4 about here

An ANOVA was also performed to compare the two groups across age, flanker type, and cue conditions in a 2 (Group: Normal vs ADHD) × 4 (Age: 6, 7, 8, and 9) × 4 (Cue type: no cue, center cue, spatial cue, and double cue) × 3 (Flanker type: congruent, incongruent, and neutral) design. This analysis showed significant main effects of group, $F(1, 2332) = 404.5$, $p < .001$, age, $F(3, 232) = 8.22$, $p < .001$, cue type, $F(3, 696) = 143.9$,

$p < .001$, and flanker type, $F(2, 464) = 404.6$, $p < .001$. Children with ADHD were slower in responding to the target (M = 1066.7 ms) compared to normal children (M = 730.1 ms). The Performance of 6 year-old children was significantly different from 8 year-old children, $F(1, 232) = 5.15$, $p < .001$, and 7 years old children was different from 9 years old children, $F(1, 232) = 3.59$, $p < .05$. Performance was significantly different among all the cue and flanker conditions. Reaction time was faster for the spatial cue (M = 867.6 ms), followed by the double cue (M = 882.5 ms), central cue (M = 899.3 ms), and no cue condition (M = 944.1 ms). Reaction time was faster for the neutral condition (M =846.4 ms), followed by congruent (M = 880.7 ms), and incongruent condition (M = 968.1 ms).

Interaction between group and cue condition was marginally significant, $F(3, 696) = 3.60$, $p = 0.093$. A difference in performance between ADHD and normal children was expected with respect to processing the cues, specifically spatial and double cue conditions. Therefore, a planned comparison between double and spatial cue was performed in both the groups. Significant difference in performance between double cue and spatial cue condition, $F(1, 696) = 6.48$, $p < .00001$, in normal group were found. However, no difference was found between these cue conditions in ADHD group. To further validate these results, an ANOVA was performed for 2 (Group: Normal vs ADHD) × 4 (Age: 6, 7, 8, and 9) × 2 (Orienting: Exogenous vs endogenous orienting). Group, $F(1, 232) = 4.33$, $p < .05$, and orienting effect was significant, $F(1, 232) = 14.12$, $p < .001$. Orienting effect was more for endogenous orienting (M = 31.5 ms) compared to exogenous orienting (M = 16.7 ms). There was significant difference in endogenous orienting between normal and ADHD children, $F(1, 232) = 5.89$, $p < .001$, normal

children were orient better to spatial cue (M = 43.1 ms) compared to ADHD children (M = 19.9 ms). There was no difference in exogenous orienting between the two groups.

Insert Figure 5a and 5b about here

Given the difference in performance between the cue conditions, we also analyzed conflict effect for each cue condition separately. Data was submitted to 2 (Group: Normal and ADHD) × 4 (age levels: 6, 7, 8, 9) between factor ANOVA. A group effect was found for all the cues except double cue, conflict effect was more for ADHD children compared to normal children for center cue, $F(1, 232) = 6.95$, $p < .01$, spatial cue, $F(1, 232) = 8.90$, $p < .01$, and no cue, $F(1, 232) = 4.31$, $p < .05$, conditions. An age effect was found only for the double cue, $F(3, 232) = 2.71$, $p < .05$. The Performance of 6 years old children was significantly different from 9 years old children, $F(1, 232) = 3.65$, $p < .05$. An interaction between group and age was marginally significant, $F(3, 232) = 2.39$, $p = .06$. Planned comparisons indicated that 7 years old ADHD children were significantly different from 9 years old ADHD children, $F(1, 232) = 4.54$, $p < .05$ There was no change in performance between 6-9 years old normal children.

Insert Figure 6a, 6b, 6c, and 6d about here

To examine, whether the attention networks were independent or not, a correlation among the three network scores in both the groups was performed once the effect of age was adjusted. Correlation was also performed for each age of both the

groups separately. In normal children, overall conflict score was significantly correlated with the alerting, r = -0.223, p < .01, and endogenous orienting score, r = 0.218, p < .01. There was a trend of an association between conflict score and exogenous orienting score, r = 0.160, p = 0.08. This effect was not found for all the age groups. Only at age 6, conflict score was significantly correlated with the endogenous orienting score, r = 0.423, p < .05, and at age 9, conflict score was significantly correlated with the alerting score, r = -0.371, p < .05. In children with ADHD, significant correlation was only observed in alerting and exogenous orienting score, r = 0.333, p < .001, which was found for ages 7, r = 0.385, p < .05, 8, r = 0.507, p < .01, and 9 years, r = 0.532, p < .01.

Delay Aversion: *%SDR and %LDR*

The analysis yielded a significant main effect of group, $F(1, 232) = 785.1$, $p < .001$. ADHD children chose small immediate rewards (M = 76.2%) over large delayed rewards more than controls (M = 23.4%). The age effect was also found to be significant, $F(3, 232) = 7.79$, $p < .001$. The performance of 6 years old children was significantly different from 8 years old children, $F(1, 232) = 5.47$, $p < .001$. One-way ANOVA with age as a between factor was performed separately for both the groups. Age effect was found in both normal, $F(3, 116) = 4.87$, $p < 0.01$, and ADHD children, $F(3, 116) = 3.96$, $p < .01$. Performance was significantly different between 6 and 8 years old normal children, $F(1, 116) = 3.97$, $p < 0.05$, and that of 7 years old was significantly different from 9 years old, $F(1, 116) = 3.60$, $p < 0.05$. In the ADHD group, performance of 6 years old was significantly different from 7 years old, $F(1, 116) = 3.61$, $p < 0.05$ and there was no difference in performance between 7 to 9 years of age. Similar results were observed with

%LDR. ADHD children chose large delayed rewards (M = 23.7%) over small short delay rewards much less than controls (M = 76.4%), $F(1, 232) = 784.0$, $p < .001$.

Insert Figure 7a and 7b about here

Discussion

We examined the developmental trajectories of control processes such as response inhibition, error monitoring, attentional disengagement, attentional networks, and motivational style in ADHD children as compared to normal children aged 6-9 years. We found that response inhibition, error monitoring, and attentional disengagement develops between 6-9 years in normal children. Age related differences in performance with respect to the control functions were not observed in ADHD children aged 6-9 years. Age related changes were also observed in the motivational style in normal children between 6-9 years of age, while it improved only between 6-7 years in ADHD children. Age related changes were also observed on conflict score under double cue conditions in ADHD children aged 7-9 years.

Development of Response Inhibition in Normal and ADHD Children

Major developments in response inhibition seem to occur between 7-8 years of age in normal children. Other developmental studies on inhibitory control have also shown significant development between 7.5-9.5 years of age followed by 9.6-11.5 years (Brocki & Bohlin, 2004). Becker et al. (1987) reported a developmental transition in inhibitory control between 6 and 8 years of age. Active development of response inhibition between

7-8 years of age is consistent with the maturational patterns of the frontal cortex, which mediates inhibitory control (Hudspeth & Pribram, 1992).

Age related improvement in inhibitory control was not observed in children with ADHD between 6-9 years of age. The immature inhibitory control in ADHD children could be related to the behavioral findings that SSRT is larger in patients with ADHD (Oosterlaan et al., 1998). It has been argued that inferior frontal cortex (IFC) is a critical brain region for the process of inhibiting an already initiated response (Aron, & Poldrack, 2005), in which structural and functional deficits were observed in ADHD (Rubia et al., 2008). Rubia et al. (2008) found that during successful inhibition ADHD children between 9-16 years of age showed reduced activation in the left dorsolateral/inferior prefrontal cortex. Immature inhibitory control in young ADHD children is also supported by EEG studies. Spronk et al. (2008) investigated ERP measures of conflict monitoring and inhibition (Nogo-N2 and Nogo-P3), cue-orientation and pre-stimulus target expectation (Cue-P2 and P3) for 5-7 years old children with and without ADHD. They found that ADHD children detected fewer targets and had higher inattention scores accompanied by reduced centro-parietal Cue- and Go-P3 activity and reduced Nogo-P3 at fronto-central leads, which indicates early signs of delayed attention development and immature inhibitory processing between 5-7 years of age in ADHD children.

Development of Error Monitoring in Normal and ADHD Children

Age related improvements in error monitoring were observed between 6-8 years of age in normal children. This is consistent with another study in which we examined development of error monitoring in normal children with larger age group of 6-11 years

(Gupta et al., submitted, a). We found that a major development in error monitoring as measured by PES takes place between 6-10 years of age with an initial increase in PES followed by subsequent decrease indicating a curvilinear relationship between PES and age. PES was not found to uniformly decrease across the age range of 7-10 years as the decrease in PES was more substantial between 9-10 years as compared to 7-8 years of age. Together, these findings suggested that children are able to recognize the errors and are able to adjust their performance after an error. EEG/ERP studies have also reported that ERN amplitude increased with age and children with 7-12 years of age are able to consciously recognize the errors and are able to adjust their performance after an error (Davis et al., 2004).

So far, no study has closely examined the development of error monitoring using PES in children between 6-9 years, which is a period of major development of executive functions in children. A few ERP studies have briefly discussed the behavioral component (PES) of error monitoring in their studies (Wiersema et al., 2007; Davis et al., 2004; Hogan et al., 2005). For example, Wiersema et al. (2007) examined the development trajectory of error monitoring in children (aged 7-8), young adolescent (13-14), and adults (age 23-24). They found no difference between age groups with respect to PES. Davis et al. (2004) also reported no difference between age groups for PES. In contrast, Hogan et al. (2005) observed an increase in the amount of PES from adolescence (age 12-19) to adulthood (age 19-22). The diverging results could be due to combining wide age groups and difference in task requirements. The complexity of the task may also play a critical role in highlighting developmental effects in error monitoring.

Our results extend the findings of Schachar et al. (2004) showing that in addition to lesser extent of slowing, error monitoring also does not show age related improvement in ADHD children between 6-9 years of age. An immature control of error monitoring in ADHD children aged 6-9 years is consistent with the ERP correlates of error monitoring. Reduced PES was accompanied by smaller ERN (Lioti et al., 2005) and *Pe* (Wiersema et al., 2005) in ADHD children between 9-11 years. Thus, ADHD children might show deficiencies in both unconscious and conscious detections of errors between 9-11 years of age. Rubia et al. (2008) reported that children with ADHD showed reduced activation in the posterior cingulate gyrus (error monitoring network), which is implicated in error detection and subsequent enhanced functions of arousal, attention allocation, and performance monitoring that are necessary to avoid future mistakes.

Development of Attentional Disengagement in Normal and ADHD Children

Attentional disengagement/task switching also follows different developmental patterns for normal and ADHD children. We found that the overall switch cost reduced from 7-9 years of age in normal children, indicating significant development in executive control processes during this period. This is consistent with another study in which we explored the possibility of shared mechanisms underlying task switching and error monitoring through a developmental framework in the age group of 6-11 years (Gupta et al., submitted, a). We found that overall switch costs reduced from 7-10 years of age. There was no difference in task switching between 10 and 11 years of age. Task switching and error monitoring may share a common mechanism such as response inhibition. This is further supported by neuropsychological and neuroimaging studies, which have reported

the role of inferior frontal regions in switching between stimuli (Jemel et al., 2002) also found to be involved in inhibitory control (Rubia et al., 2008).

Age related differences in attentional disengagement were not observed in ADHD children between 6-9 years of age. Lack of age related improvement in attentional disengagement in ADHD children may be related to the finding of reduced dopamine activity during task switching in children with ADHD (Smith et al., 2006). Dopamine is known to be sub-optimally active in ADHD patients (Oades, 2006).

Development of Attentional Networks in Normal and ADHD Children

Age related changes were not observed in ADHD children between 6-9 years of age in an alerting network. Both the groups were different at 9 years of age relative to earlier ages which suggests a late development of alerting network in normal children. An alerting network of attention is not yet mature in ADHD children aged 9 years. Previous studies have also suggested a slow development of alerting network in normal children (Ridderinkhof et al., 1997). Rueda et al. (2004) observed stability in middle childhood (6-9 years) and found some improvement in late childhood (10 years) in the alerting network. Delayed development of alerting network in ADHD may be a direct consequence of low levels of noradrenergic neurotransmitter (Beane & Marrocco, 2004), which was found to be critical for the alerting system (Marrocco & Davidson, 1998).

The overall conflict effect did not reduce between 6-9 years of age in both the groups. However, in the double cue condition the conflict effect was reduced between 7-9 years of age in ADHD children. It supports the previous findings that double cue can

rescue the attention deficit of the ADHD children, presumably via a phasic increase in alertness. Children with ADHD made less omission errors in the double cue condition compared to no cue and center cue conditions (Johnson et al., 2008). Posner et al. (1990) reported that patients with frontal lesions were slow to initiate responses when a target stimulus was not preceded by a warning cue, relative to when this cue was present. These findings indicate a problem with the 'tonic' or internal aspects of alertness but an intact ability to use cues to improve performance. Tonic levels of alertness are thought to be modulated by noradrenaline and difficulties with alertness might arise due to deficient fronto-parietal control over the locus coeruleus (Halperin & Schulz, 2006). These results are consistent with the current theories of ADHD, which emphasize a problem of regulation of arousal in ADHD (Johnson et al., 2007). Children with ADHD may find it more difficult to regulate their arousal in the absence of an alerting cue than when it is present.

We did not find any age related improvement in exogenous and endogenous orienting between 6-9 years of age in both the groups, which is consistent with previous literature (Rueda et al., 2004). Early developmental changes occurred in the orienting network. ADHD children were impaired in endogenous orienting and had intact exogenous orienting (Jonkman, 2005). Carter et al. (1995) found that anticipation error was higher in ADHD children aged between 9-12 years only in an endogenous cue condition, hence, the tendency to make more anticipation (impulsive) errors when stimulus appearance was predictable.

Interaction among the Four Networks in Children

Interaction among the attentional networks was observed in the present study, which was not consistent with a previous study (Rueda et al., 2004). It was reported that children of 7 years of age showed independence among the three networks. This difference could be attributed to difference in sample size, as the number of participants was fewer in their study (N = 44) compared to the present study (N = 120). We observed a developmental shift in the interaction among the attentional networks, which may be related to the progressive formation of neural circuits. Akhtar and Enns (1989**)** also found an interaction between orienting and conflict effect that reduced from 5 years of age through adulthood. In children with ADHD, the alerting score was found to be correlated with the exogenous orienting score and conflict score of the double cue condition, which further strengthens the finding that double cue that can rescue the attention deficit of the ADHD children, presumably via a phasic increase in alertness (Johnson et al., 2008).

Development of motivational style in Normal and ADHD Children

We found that the tendency to avoid delay improved between 6-9 years of age in normal children while it improved between 6-7 years of age in ADHD children and did not improved between 7-9 years. Our results extend the findings of delay aversion theory (Sonuga-Barke, 2002) showing that ADHD children usually prefer not to wait, which did not change between 7-9 years of age.

Data showed different developmental trajectories depending on the type of control functions. In normal children, major development in response inhibition was observed between 7-8 years, error monitoring in 6-9 years, attentional disengagement in 7-9 years,

and delay aversion in 6-9 years. Late development in alerting network was observed at 9 years of age in normal children. It appears that 6-9 years is a critical period showing developmental changes in most of the cognitive functions. Therefore, it is possible that development of one function may affect the development of another function between 6-9 years of age. For example, it has been reported that the anterior system mediates executive functions such as developing and maintaining expectations (Carr, 1992), which may help in performing the tests of executive attention such as Stop Signal Task. ADHD children performed poorly on Stop Signal Task that may be because of the problem in endogenous attentional system in ADHD children. In addition, we also found that the extent of slowing after failed inhibition error was less in ADHD children compared to normal children, which may be because of a motivation to avoid delay found in ADHD children. Comparator theories of error monitoring suggested that slowing after an error may result from a comparison of the representation of recently executed responses with memory of the instruction set for the task (Scheffers & Coles, 2000). This comparison process takes time and delays the response on subsequent trials. Therefore, in order to avoid delay, ADHD children may not be willing to compare the executed responses with memory of the instruction set of the task and hence do not show subsequent behavioural adjustment following an error. Thus, no change with age in delay aversion may hinder the development of error monitoring in ADHD children.

Our results also throw some light on the long lasting debate of unitary and component views of executive control. Unitary theory of executive control posits a unified mechanism or a common resource underlying various aspects of executive control. The component theories of executive control argue that executive control

consists of a number of distinct components such as response inhibition, error monitoring, attentional disengagement, delay aversion, task switching, and error monitoring. The results of the present study support the component theory of executive control. Different developmental trajectories observed for each of the control functions, indicate different mechanisms underlying the control functions. These findings also have implications for the theory of ADHD. Barkley (1997) argued that response inhibition is the primary deficit found in ADHD, which in turn affects the other executive functions such as working memory, error monitoring and so on. If this is the case then response inhibition should develop earlier than error monitoring. However, we found developmental changes in response inhibition between 7-8 years of age while for error monitoring between 6-8 years of age, which indicates an early development of error monitoring as compared to response inhibition. Posner and Rothbart (1998) also found that the ability to detect an error (indicated by PES) develops at an earlier age than the ability to inhibit. Further studies on the relationship between inhibition and executive functions are needed to determine whether there is a need to modify Barkley's model to account for the component view of executive functions rather than a unified perspective to executive function deficit as the underlying mechanism for ADHD.

In addition, because of smaller age range of children in the present study, we can not comment on whether ADHD could involve a developmental delay or a stable deficit in control processes. However, the developmental pattern obtained in the present study favors the conceptual view of ADHD as a stable deficit in cognitive control functions. For example, performance of 9 years old ADHD children was poor from the performance of 6 years normal children on all the tasks. These results are consistent with studies that

report a deficit in control functions as response inhibition, error monitoring, attentional disengagement, attentional networks, and motivational style in adults with ADHD (Aron et al., 2003; O' Connell et al., 2009; Tucha et al., 2005; Oberlin et al., 2005; Plichta et al., 2009). Results of the present study should be further validated with larger age group of children with a longitudinal design on both structural as well as functional brain development. This would inform whether delay in structural brain development can be mapped onto the delay in functional brain and cognitive development in ADHD.

Summary and Conclusions

Mapping of the developmental trajectories of various control processes such as response inhibition, error monitoring, attentional disengagement, attentional networks, and motivational style, from 6-9 years of age is important in understanding the pathology of ADHD. We demonstrate the period of 6-9 years to be developmentally active with respect to the control processes in normal children but not in children with ADHD. Since the difference across the age range of 6 to 9 years in ADHD children was not significant it appears that the deficits in control processes accumulate with age. The developmental patterns of the control processes in normal children supports the component view of executive control. Present study favors the conceptual view of ADHD as a stable deficit in cognitive control functions rather than developmental delay in control functions, which are implicated in the pathology of ADHD. Such developmental trends of control functions in ADHD are consistent with a multi-factorial cognitive etiology model of ADHD. In this model, an impulsive cognitive style is attributed to an additive or interactive dysfunction in multiple cognitive systems and their closely related mediating neural networks (Willcutt et al., 2005). Developmental trajectories of these processes in

late childhood could be further examined, which is also marked by the relative improvement in symptoms of ADHD.

References

1. Akhtar, N., & Enns, J. T. (1989). Relations between covert orienting and filtering in the development of visual attention. *Journal of Experimental Child Psychology, 48,* 315-344.
2. American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.
3. 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,* 1285-1292.
4. Aron, A. R., Dowson, J. H., Sahakian, B. J., & Robbins, T. W. (2003). Methylphenidate improves response inhibition in adults with attention-deficit/hyperactivity disorder. *Biological Psychiatry, 54,* 1465-1468.
5. Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of AD/HD. *Psychological Bulletin, 121,* 65-94.
6. Beane, M., & Marrocco, R.T. (2004). Norepinephrine and acetylcholine mediation of the components of reflexive attention: implications for attention deficit disorders. *Progress in Neurobiology, 74,* 167-181.
7. Becker, M. G., Isaac, W., & Hynd, G. W. (1987). Neuropsychological development of nonverbal behaviors attributed to 'frontal lobe' functioning. *Developmental Neuropsychology, 3,* 275–298.
8. Blane, M., & Marrocco, R. (2004). Cholinergic and Noradrenergic inputs to the posterior parietal cortex modulate the components of exogenous attention. In M.I.

Posner (Ed.), Cognitive neuroscience of attention (pp. 313-325). New York: Guilford.

9. Brocki, K. C., & Bohlin, G. (2004). Executive functions in children aged 6 to 13: A dimensional and developmental study. *Developmental Neuropsychology, 26,* 571-593.
10. Brocki, K. C., & Bohlin, G. (2006). Developmental changes in the relation between executive functions and symptoms of ADHD and co-occuring behaviour problems. *Infant and Child Development, 15,* 19-40.
11. Brodeur, D. A., & Pond, M. (2001). The Development of Selective Attention in Children With Attention Deficit Hyperactivity Disorder - Statistical Data Included. *Journal of Abnormal Child Psychology, 29,* 229-239.
12. Bunge, S. A., Dudukovic, N. M., Thomason, M. E., Vaidya, C. J., & Gabrieli, J. D. E. (2002). Immature frontal lobe contributions to cognitive control in children: evidence from fMRI. *Neuron, 33,* 301-311.
13. Carr, T. H. (1992). Automaticity and cognitive anatomy: Is word recognition automatic? *American Journal of Psychology, 105,* 201-237.
14. Carter, C. S., Krener, P., Chaderjian, M., Northcutt, C., & Wolfe, V. (1995). Assymetrical visual-spatial attentional performance in ADHD: evidence for a right hemisphere deficit. *Biological Psychiatry, 37,* 789-797.
15. Cepeda, N. J., Cepeda, M. L., & Kramer, A. F. (2000). Task Switching and Attention Deficit Hyperactivity Disorder. *Journal of Abnormal Child Psychology, 28,* 213-226.

16. Cepeda, N. J., Kramer, A. F., & Gonzalez de Sather, J. C. M. G. (2001). Changes in executive control across the life span: examination of task-switching performance. *Developmental Psychology, 37,* 715-730.
17. Christ, S. E., White, D. A., Mandernach, T., Keys, B. A. (2001). Inhibitory Control across the Life Span. *Developmental Neuropsychology, 20*, 653-669.
18. Conners, C. K. (2002). *Manual for Conners' rating scales*. Revised ed. N. Tonoawanda, NY: Multi-Health Systems Inc.
19. Crone, E. A., Bunge, S. A., van der Molen, M. W., & Ridderinkhof, K. R. (2006). Switching between tasks and responses: a developmental study. *Developmental Science, 9,* 278-287.
20. Davies, P. L., Segalowitz, S. L., Gavin, W. J. (2004). Development of Response-Monitoring ERPs in 7-to 25-year-Olds. *Developmental Neuropsychology, 25,* 355-376.
21. Gerstadt, C. L., Hong, Y. J., & Diamond, A. (1994). The relationship between cognition and action: performance of $3^1/_2$ years old on a Stroop-like day-night test. *Cognition, 53*, 129-153.
22. Gupta, R., Kar, B. R., & Srinivasan, N. (submitted, a). Development of Task switching and Error Monitoring in Children.
23. Gupta, R., Kar, B. R., & Srinivasan, N. (submitted, b). Cognitive Markers of ADHD: Development of a Diagnostic System.
24. Gupta, R., Kar, B. R., & Thapa, K. (2006). Specific Cognitive Dysfunction in ADHD: An Overview. In J. Mukherjee & V. Prakash (Eds.), *Recent Developments in Psychology*, pp. 153-170, Delhi.

25. Halperin, J. M., & Schulz, K. P. (2006). Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. *Psychological Bulletin, 132*, 560-581.
26. Hogan, A. M., Vargha-Khadem, F., Kirkham, F. J., & Baldeweg, T. (2005). Maturation of action monitoring from adolescence to adulthood: an ERP study. *Developmental Science, 8,* 525-534.
27. Hudspeth, W. J., & Pribram, K. H. (1992). Psychophysiological indices of cerebral maturation. *International Journal of Psychophysiology, 12,* 19-29.
28. Iaboni, F., Douglas, V. I., & Baker, A. G. (1995). Effects of reward and response cost on inhibition in ADHD children. *Journal of Abnormal Psychology, 104,* 232-240.
29. Jemel, B., Achenbach, C., Muiller, B., Ropcke, B., & Oades, R. D. (2002). Mismatch negativity results from bilateral asymmetric dipole sources in the frontal and temporal lobes. *Brain Topography, 15,* 13-27.
30. Johnson, K. A., Kelly, S. P., Bellgrove, M. A., Barry, E., Cox, E., Gill, M., et al. (2007). Response variability in attention deficit hyperactivity disorder: Evidence for neuropsychological heterogeneity. *Neuropsychologia, 45,* 630-638.
31. Johnson, K. A., Robertson, I. H., Barry, E., Mulligan, A., Daibhis, A., Daly, M., Watchorn, A., Gill, M., & Bellgrove, M. A. (2008). Impaired conflict resolution and alerting in children with ADHD: evidence from the Attention Network Task (ANT). *The Journal of Child Psychology and Psychiatry, 49,* 1339-1347.

32. Johnstone, S. J., Carly, B. P., Robert, J. B., Adam, R. C., & Janette, L. S. (2005). Development of Inhibitory processing During the Go/NoGo Task. *Journal of Psychophysiology, 19*, 11-23.
33. Jonkman, L. M. (2005). Selective Attention Deficits in Children With Attention Deficit Hyperactivity Disorder: A Review of Behavioral and Electrophysiological Studies. In D. Gozal D. L. Molfese (Eds.), *Attention Deficit Hyperactivity Disorder: From Genes to Patients.* Totowa, NJ, pp. 255-274).
34. Kramer, A. F., Humphrey, D. G., Larish, J. F., Logan, G. D., & Strayer, D. L. (1994). Aging and inhibition: Beyond a unitary view of inhibition processing in attention. *Psychology of Aging, 9*, 491-512.
35. Liotti, M., Pliszka, S. R., Perez, R., Kothmann, D., & Woldorff, M. G. (2005). Abnormal brain activity related to performance monitoring and error detection in children with ADHD. *Cortex, 41*, 377-388.
36. Logan, G. D. (1994). On the ability to inhibit thought and action. A users' guide to the stop signal paradigm. In: Dagenbach, D., & Carr, T. H. (Eds.*). Inhibitory processes in attention, memory and language,* San Diego (CA): Academic Press. 189–236.
37. Marrocco, R. T., & Davidson, M. C. (1998). Neurochemistry of attention. In: Parasuraman, R. (Ed). *The Attentive Brain*. Cambridge University Press, Cambridge.
38. Monsell, S. (2003). Task Switching. *Trends in Cognitive Science, 7***,** 134-140.
39. O' Connell, R. G., Bellqrove, M. A., Dockree, P. M., Lau, A., Hester, R., Garavan, H., Fitzqerald, M., Foxe, J. J., & Robertson, I. H. (2009). The neural

correlates of deficient error awareness in attention-deficit hyperactivity disorder (ADHD). *Neuropsychologia, 47*, 1149-1159.

40. Oades, R. D. (2006). Function and dysfunction of monoamine interactions in children and adolescents with AD/HD in E. D. Levin (Ed.), *Neurotransmitter interactions and cognitive function* (pp. 207-244). Basel: Birkhauser Verlag.
41. Oberlin, B. G., Alford, J. L., & Marrocco, R. T. (2005). Normal attention orienting but abnormal stimulus alerting and conflict effect in coombined subtype of ADHD. *Behavioural Brain Research, 165,* 1-11.
42. Oosterlaan, J., & Sergeant, J.A. (1998). Response inhibition and response re-engagement in attention deficit/hyperactivity disorder, disruptive, anxious and normal children. *Behavioral Brain Research*, 94, 33-43.
43. Oosterlaan, J., Logan, G. D., & Sergeant, J.A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD+CD, anxious, and control children: a meta-analysis of studies with the stop task. *Journal of Child Psychology and Psychiatry, 39,* 411-425.
44. Pennington, B.F., & Ozonoff, S. (1996). Executive functions and development of psychopathology. *Journal of Child psychology and Psychiatry and Allied Disciplines, 37,* 51-87.
45. Plichta, M. M., Vasic, N., Wolf, R. C., Lesch, K. P., Brummer, D., Jacob, C., Fallgatter, A. J., & Garon, G. (2009). Neural hyporesponsiveness and hyperresponsiveness during immediate and delayed reward processing in adult attention-deficit/hyperactivity disorder. *Biological Psychiatry, 65,* 5-6.

46. Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13*, 25-42.

47. Posner, M. I., Rothbart, M. K. (1998). Attention, self-regulation, and consciousness. *Philosophical Transactions of the Royal Society of London, B, 353*, 1915-1927.

48. Rabbit, P. M. A. (1966). Error correction time without external error signals. *Nature, 212*, 438.

49. Raven, J., Raven, J. C., & Court, J. H. (1998). *Colored Progressive Matrices*. Oxford: Oxford Psychologists Press.

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

51. Rubia, K., Halari, R., Smith, A. B., Mohammed, M., Scott, S., Giampietro, V., Taylor, E., & Brammer, M. J. (2008). Dissociated Functional Brain Abnormalities of Inhibition in Boys With Pure Conduct Disorder an din Boys With Pure Attention Deficit Hyperactivity Disorder. *American Journal of Psychiatry, 165*, 889-897.

52. Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., & Posner, M. I. (2004a). Development of attentional networks in childhood. *Neuropsychologia, 42*, 1029-1040.

53. Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., & McCandlis, B. D., et al. (2004b). Development of attention during childhood. *Neuropsychologia, 42*, 1029-1040.

54. Schachar, R. J., Chen, S., Logan, G. D., Ornstein, T. J., Crosbie, J., Ickowicz, A., & Pakulak, A. (2004). Evidence for an Error Monitoring Deficit in Attention Deficit Hyperactivity Disorder. *Journal of Abnormal Child Psychology, 32,* 285-293.

55. Scheffers, M. K., & Coles, M. G. (2000). Performance monitoring in a confusing world: Error related brain activity, judgments of response accuracy, and types of errors. *Journal of Experimental Psychology: Human Perception and Performance,* 26, 141-151.

56. Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., Clasen, L., Evans, A., Giedd, J., & Rapoport, J. L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of the Sciences of the United States of America, 104,* 19649-19654.

57. Smith, A. B., Taylor, E., Brammer, M., Toone, B., & Rubia, K. (2006). Task-specific hypoactivation in prefrontal and temporoparietal brain regions during motor inhibition and task switching in medication-naïve children and adolescents with attention deficit hyperactivity disorder. *American Journal of Psychiatry, 163,* 957-960.

58. Sonuga-Barke E. J. (2002). Psychological heterogeneity in AD/HD-a dual pathway model of behavior and cognition. *Behavioral Brain Research. 130,* 29-36

59. Spieler, D. H., Balota, D. A., & Faust, M. E. (1996). Stroop performance in healthy younger and older adults and in individual with dementia of the

Alzheimer's type. *Journal of Experimental Psychology: Human Perception & Performance, 22*, 461-479.

60. Spronk, M., Jonkman, L. M., & Kemner, C. (2008). Response inhibition and attention processing in 5-to7-year old children with and without symptoms of ADHD: An ERP study. *Clinical Neurophysiology, 119*, 2738-2752.
61. Tucha, O., Mecklinger, L., Laufkotter, R., Kaunzinger, I., Paul, G. M., Klein, H. E., & Lange, K. W.(2005). Clustering and switching on verbal and figural fluency functions in adults with attention deficit hyperactivity disorder. *Cognitive Neuropsychiatry, 10*, 231-248.
62. Wiersema, J. R., van der Meere, J. J., & Roeyers, H. (2005). ERP correlates of impaired error monitoring in children with ADHD. *Journal of Neural Transmission, 112*, 1417-1430.
63. Wiersema, J. R., van der Meere, J. J., & Roeyers, H. (2007). Developmental change in error monitoring: An event-related potential study. *Neuropsychologia, 45*, 1649-1657.
64. Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington. B. F. (2005). Validity of the executive function theory of Attention Deficit/Hyperactivity Disorder: A meta-analytic review. *Biological Psychiatry, 57*, 1336-1346.
65. Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the life span. *Developmental Psychology, 35*, 205-213.

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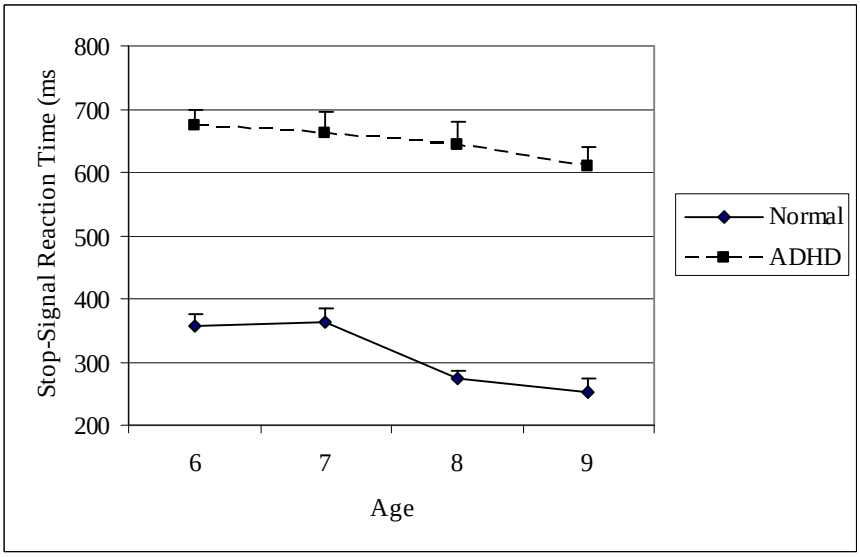
Figure 7: %SDR (a), and %LDR (b) of all the four age groups of Normal and ADHD children.

Table 1

Characteristics	ADHD (N = 120) 6-9 Years		Normal Controls (N = 120) 6-9 Years		F
	M	SD	M	SD	
Age (years)	7.78	1.13	7.80	1.11	0.62
Intellectual Ability Raw Score(Percentile)	27.2(85.2)	3.5(18.6)	28.2(88.6)	3.88(15.7)	2.24
CPRS-R-LF DSM-IV total score for ADHD (T score)	43.4(80.3)	3.7(3.7)	3.8(43.2)	1.0(1.5)	11191.75***

Note: *p < .05; **p < .01; ***p < .001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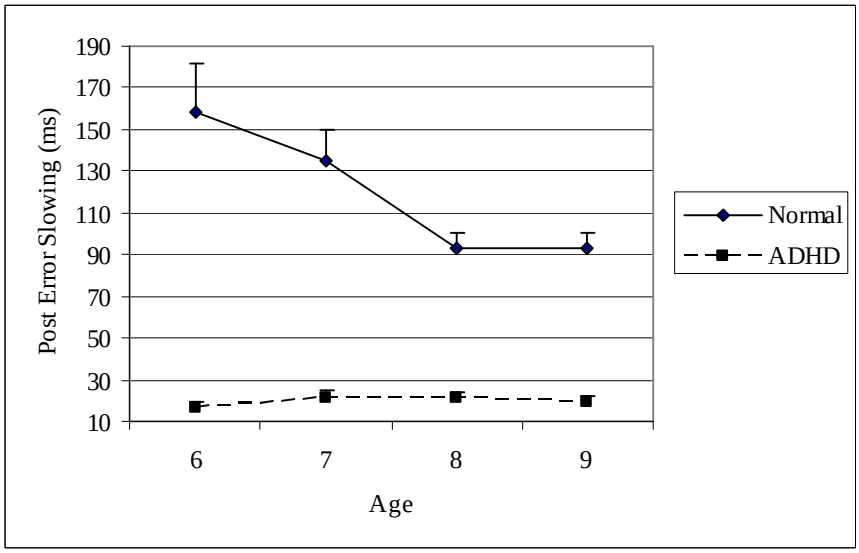
600
500
400
300
200
Stop-Signal Reaction Time (ms
6
7
8
9
Age
Normal
ADHD

Figure 1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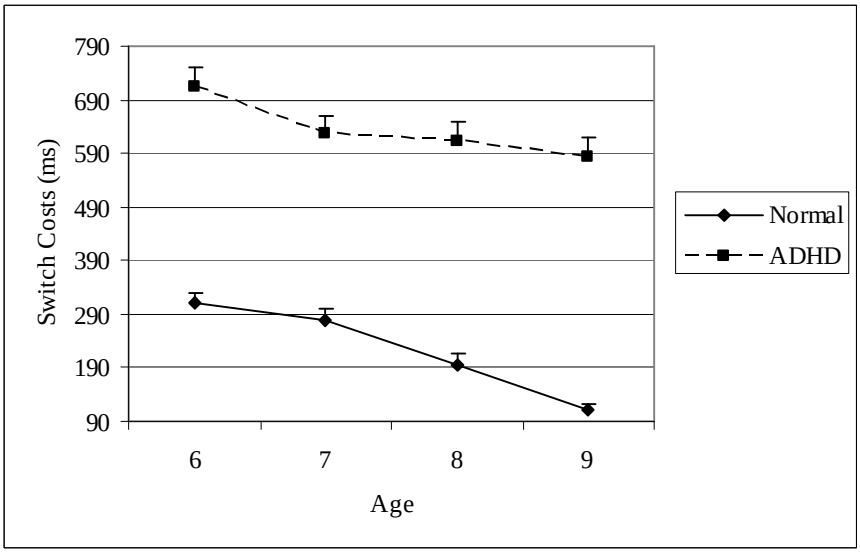
30
10
Post Error Slowing (ms)
6
7
8
9
Age
Normal
ADHD

Figure 2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Switch Costs (ms)
6
7
8
9
Age
Normal
ADHD

Figure 3

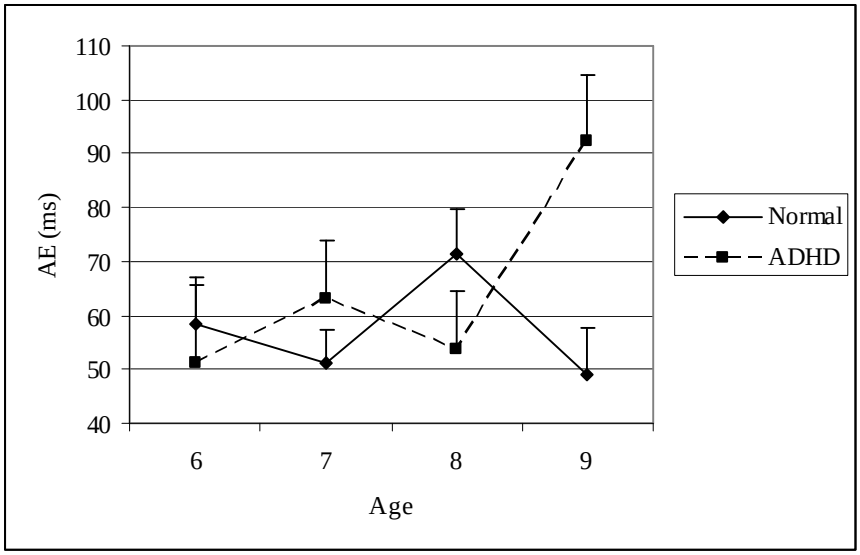


Figure 4

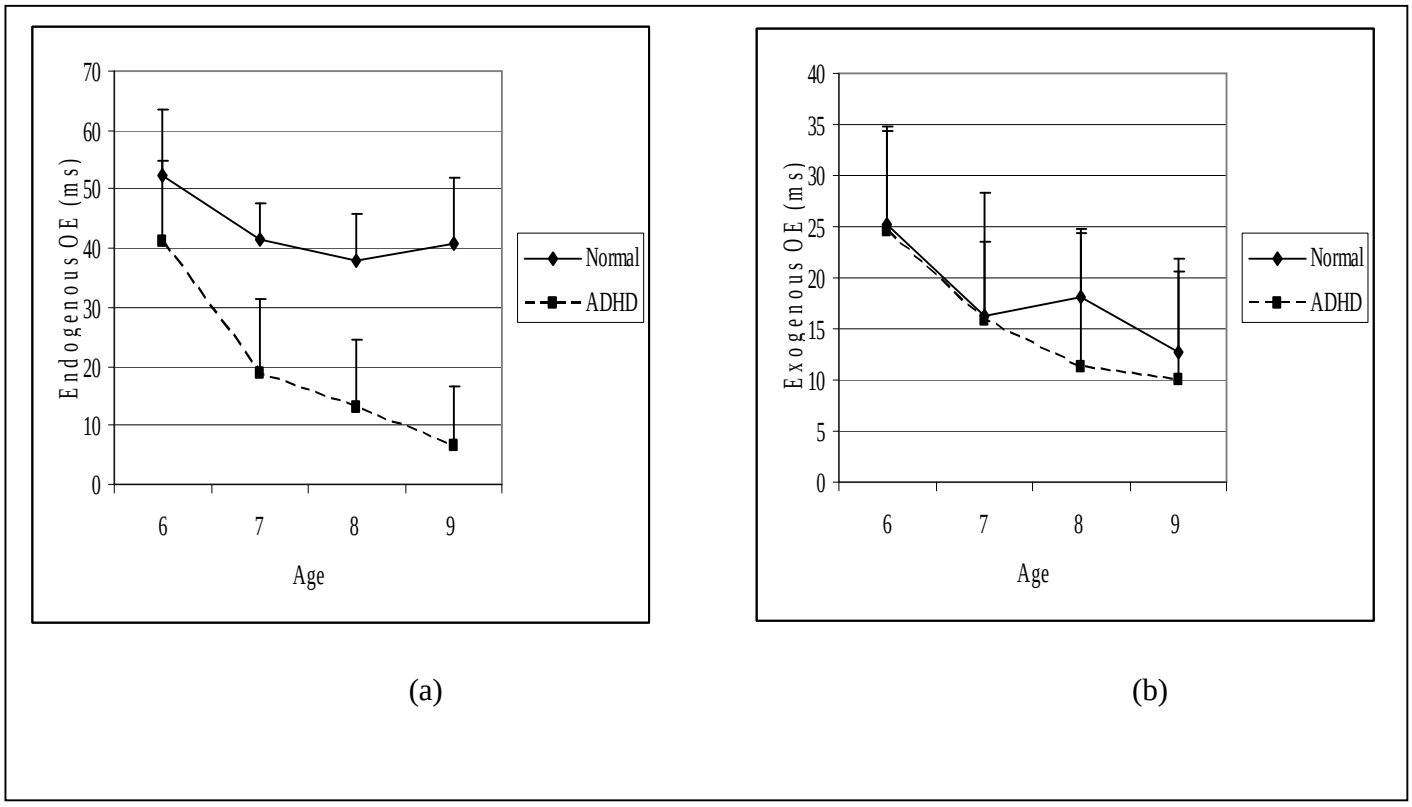

70
60
50
40
30
20
10
0
Endogenous OE (ms)
6
7
8
9
Age
Normal
ADHD
(a)
40
35
30
25
20
15
10
5
0
Exogenous OE (ms)
6
7
8
9
Age
Normal
ADHD
(b)

Figure 5

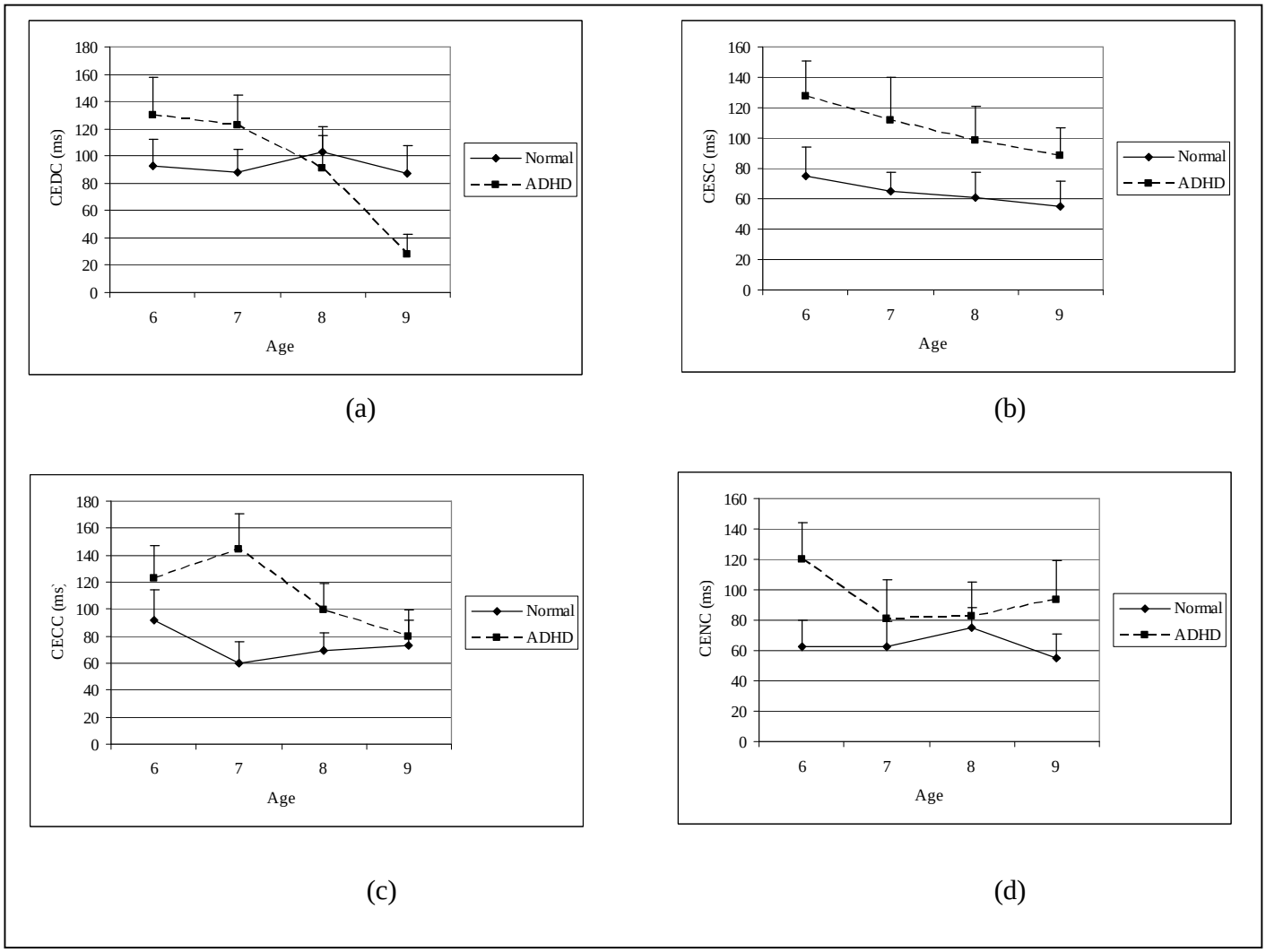
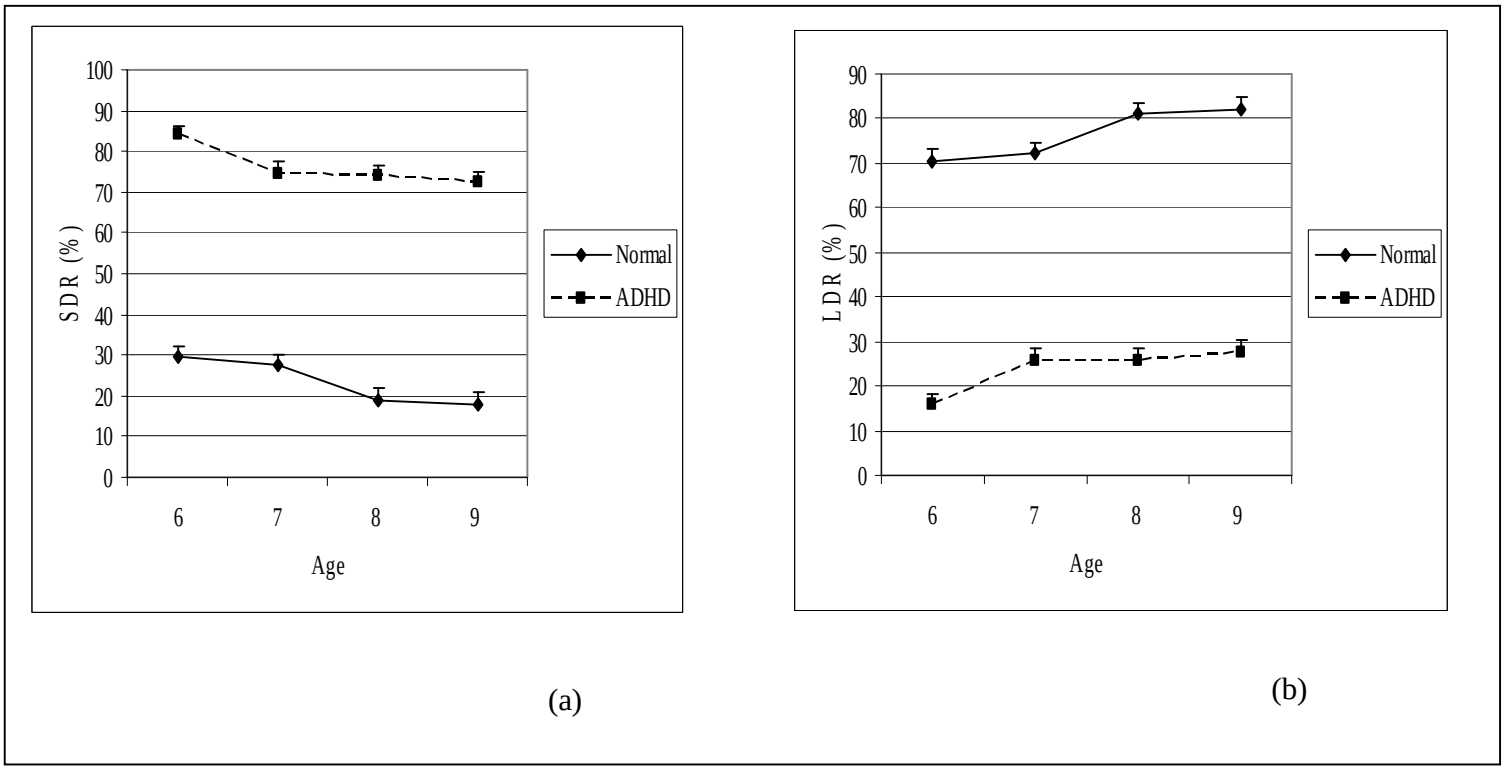

CEDC (ms)
180
160
140
120
100
80
60
40
20
0
6
7
8
9
Age
Normal
ADHD
(a)
CESC (ms)
160
140
120
100
80
60
40
20
0
6
7
8
9
Age
Normal
ADHD
(b)
CECC (ms)
180
160
140
120
100
80
60
40
20
0
6
7
8
9
Age
Normal
ADHD
(c)
CENC (ms)
160
140
120
100
80
60
40
20
0
6
7
8
9
Age
Normal
ADHD
(d)

Figure 6


100
90
80
70
60
50
40
30
20
10
0
SDR (%)
6
7
8
9
Age
Normal
ADHD
90
80
70
60
50
40
30
20
10
0
LDR (%)
6
7
8
9
Age
Normal
ADHD

(a) (b)

Figure 7