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Heart rate and reinforcement sensitivity in ADHD

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Background: Both theoretical and clinical accounts of attention-deficit/hyperactivity disorder (ADHD) implicate a dysfunctional reinforcement system. This study investigated heart rate parameters in response to feedback associated with reward and response cost in ADHD children and controls aged 8 to 12. **Methods:** Heart rate responses (HRRs) following feedback and heart rate variability (HRV) in the low frequency band (.04–.08 Hz), a measure of mental effort, were calculated during a time production paradigm. Performance was coupled to monetary gain, loss or feedback-only in a cross-over design. **Results:** Children with ADHD exhibited smaller HRRs to feedback compared to controls. HRV of children with ADHD decreased when performance was coupled to reward or response cost compared to feedback-only. HRV of controls was similar across conditions. **Conclusions:** Children with ADHD were characterised by (a) possible abnormalities in feedback monitoring and (b) motivational deficits, when no external reinforcement is present. **Keywords:** ADHD, reinforcement, feedback, motivation, task engagement, heart rate. **Abbreviations:** DBD: disruptive behaviour disorder rating scale; HRR: heart rate response; HRV: heart rate variability; IBI: inter-beat interval.

Attention deficit hyperactivity disorder (ADHD) is characterised by inattention, impulsive behaviour and motor restlessness. One of the candidate endophenotypes of ADHD is a dysfunctional reinforcement system (Castellanos & Tannock, 2002; Doyle et al., 2005; Sagvolden, Johansen, Aase, & Russell, 2005; Sonuga-Barke, 2002) that is responsible for cognitive and motor limitations in ADHD. A review of 21 studies on the impact of reinforcement in ADHD revealed that the improvement in task performance by using appropriate reinforcement was somewhat larger in ADHD children than in controls (Luman, Oosterlaan, & Sergeant, 2005). ADHD is considered a motivational problem whereby ADHD individuals are unable to show optimal performance by using intrinsic motivation (Douglas, 1989; Sergeant, Oosterlaan, & Van der Meere, 1999). Others stress dysfunctions in processing stimuli that are related to reward and extinction, caused by fronto-limbic abnormalities in ADHD (Sagvolden et al., 2005; Sonuga-Barke, 2002). The goal of this study was to seek psychobiological evidence for an abnormal reinforcement sensitivity and motivational deficits in ADHD by measuring (a) the short-term change in heart rate following reinforcement feedback (heart rate response, HRR) and (b) heart rate variability (HRV) under different reinforcement conditions. These measures are both sensitive to reinforcement processing (Crone, Bunge, De Klerk, & Van der Molen, 2005; Suess, Newlin, & Porges, 1997).

Experimental studies of ADHD children have identified abnormalities in self-rated and observed motivation (Carlson, Booth, Shin, & Canu, 2002), in both cognitive (Slusarek, Velling, Bunk, & Eggers, 2001) and academic performance (Volkow et al., 2004). Otherwise, children with ADHD demonstrate dysfunctions in several aspects of (reinforcement) feedback processing such as: error detection (internal detection of whether a response was correct) (Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005), error appraisal (Wiersema, Van der Meere, & Roeyers, 2005), reinforcement processing (Van Meel, Oosterlaan, Heslenfeld, & Sergeant, 2005a) and error-related compensatory responses such as post-error slowing (Schachar et al., 2004; Sergeant & Van der Meere, 1988). By adding reinforcement to feedback, performance of ADHD children may improve by shaping behaviour (Keitz, Martin-Soelch & Leenders, 2003; Schultz, 2000). Alternatively, performance may improve by an increase in motivation (Cameron & Pierce, 1994).

When humans receive feedback, heart rate is found to decelerate (Somsen, Van der Molen, Jennings, & Van Beek, 2000; Van der Veen, Van der Molen, Crone, & Jennings, 2004). This deceleration is larger following negative than positive feedback (Somsen et al., 2000). The degree of cardiac deceleration is dependent, firstly, on the information value regarding performance and secondly, on the mismatch between the feedback expectation and the actual feedback (Somsen et al., 2000). When the mismatch between expected and actual feedback is large, heart rate decelerates. Crone et al. (2005) demonstrated that heart rate deceleration was larger when the financial gain that was related to feedback increased.

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Variability in heart rate (HRV) is considered an indicator of motivation, since tasks that demand cognitive effort or active attention evoke lower HRV (see for a review Jorna, 1992). Three cardiac rhythmicities have been identified (Hyde & Izard, 1997). The fastest rhythmicity (.15–.60 Hz) is the respiratory sinus arrhythmia (RSA), which reflects synchronisation of the heart with the respiratory system. Higher RSA in children has been associated with attention to novel stimuli (Porges, 1991). The intermediate frequency (.08–.15 Hz), the Traube–Herring–Meyer (THM) wave, reflects synchronisation of the heart with blood pressure and the slowest rhythm (.04–.08 Hz), the angiotensin-renin vasomotor (ARV) rhythmicity, reflects synchronisation of the heart with the peripheral vascular system. Diminished variability in the THM and, more specifically, variability in the ARV rhythm are associated with sustained mental effort in adults (Mulder & Mulder, 1981) and in children (Hyde & Izard, 1997). Interestingly, experimental studies indicate that both motivation and reward reduce HRV (Pruyn, Aasman, & Wyers, 1985; Suess, Newlin, & Porges, 1997), while time-on-task (diminished allocation of effort) increases HRV.

Decreased HRRs to reinforcement feedback in ADHD have been reported (Crone, Jennings, & Van der Molen, 2003; Iaboni, Douglas, & Ditto, 1997). Both Iaboni et al. (1997) and Crone et al. (2003) demonstrated that the differential effects of positive and negative reinforcement were less pronounced for HRR in ADHD children than in controls. Increased HRV within the middle frequency band (.07–.15) has been reported in ADHD children compared to controls, indicating motivational problems in ADHD children (Börger et al., 1999; Börger & Van der Meere, 2000). This finding is consistent with current theoretical frameworks claiming that ADHD children have difficulties maintaining an optimal intrinsic level of motivation (e.g., Sergeant et al., 1999).

We used three conditions to study the impact of reinforcement feedback on performance: (1) feedback-only, (2) reward for correct responses, (3) response cost for incorrect responses. Since there is evidence of diminished feedback processing in ADHD (Van Meel et al., 2005a), we expect the HRR to reinforcement feedback to be less pronounced in children with ADHD than in controls. When both the reward and extinction systems are dysfunctional in ADHD (Sagvolden et al., 2005), the increase in HRR when reinforcement is added to feedback (Crone et al., 2005) is expected to be smaller in children with ADHD than in controls.

It is suggested that children with ADHD suffer from motivational problems (Douglas, 1989; Sergeant et al., 1999) and therefore, HRV is predicted to be larger (lower task engagement) in children with ADHD than in controls. In the reinforcement conditions compared to the feedback-only, the group difference in HRV is expected to decrease, since reinforcement increases motivation (Cameron & Pierce, 1994) and lowers HRV (Suess, Newlin, & Porges, 1997).

Method

Participants

Eighteen children aged 8 to 12 (three girls; mean age 122 months) with a clinical diagnosis of ADHD were compared to 18 age-matched normal control children (two girls, mean age 124 months). All parents completed a written informed consent prior to the experiment and the experiment was approved by the ethics committee of the Vrije Universiteit Amsterdam. Inclusion criteria were the following: (1) Intelligence quotient >70, estimated by four subtests of the Revised Wechsler Intelligence Scale for Children: Block Design, Picture Arrangement, Arithmetic and Vocabulary (Groth-Marnat, 1997), (2) absence of any neurological disorders, learning disabilities, sensory or motor impairment, and (3) absence of all psychiatric disorders other than oppositional defiant disorder (ODD) or conduct disorder (CD) for children with ADHD, absence of all psychiatric disorders including ADHD, ODD or CD for normal controls. Background information on the participants is presented in Table 1.

The ADHD group was recruited via the Dutch ADHD parent association and all children in this group had a clinical diagnosis of ADHD. A structured clinical

Table 1 Background and clinical characteristics of the ADHD and control group

	Group				
	ADHD ($n = 18$)		Normal controls ($n = 18$)		
Measure	Mean	SD	Mean	SD	F1, 34
No. of males	15	–	16	–	–
Age in months	122	14.7	124	15.6	ns
Estimated full scale IQ	93.4	14.1	112.3	12.4	14.3*
DBD parents					
Inattention	20.8[a]	4.1	2.2	2.4	273.4**
Hyperactivity/ Impulsivity	17.8[a]	7.7	2.1	2.1	70.4**
ODD	9.7	5.5	1.4	1.5	36.2**
CD	2.7	2.1	.1	.2	82.4**
DBD teacher					
Inattention	16.3[a]	5.6	1.8	3.9	71.6**
Hyperactivity/ Impulsivity	14.9[a]	5.3	1.7	2.8	82.2**
ODD	7.8	5.1	.3	1.0	32.6**
CD	2.6	3.6	.1	.2	8.9*
FSSC-R (decile scores)	4.3	.7	6.2	.6	ns[b]

Note: ADHD = attention–deficit–hyperactivity disorder, CD = conduct disorder, DBD = Disrupted Behavior Disorder Subscale, FSSCR = revised Fear Survey Schedule for Children, ODD = oppositional defiant disorder. [a]Clinical score (>95th percentile); [b]Data of two children were missing (one ADHD, one control).
*$p < .01$, **$p < .001$, ns = $p > .05$.

interview (Diagnostic Interview Schedule for Children, based on the DSM-IV, DISC-IV) was administered to the parents of ADHD children to confirm the diagnosis (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). Sixteen children met DISC-IV criteria for ADHD combined subtype and two other children for the inattentive subtype. Eleven children met the criteria for an additional ODD diagnosis and two children were comorbid for CD. The Disruptive Behaviour Disorder rating scale (DBD, parent and teacher version) served to confirm the pervasiveness of ADHD symptoms (Pelham, Gnagy, Greenslade, & Milich, 1992). Children in the ADHD group were required to score within the clinical range (95th to 100th percentile) on both the parent and teacher DBD for either the Inattention or Hyperactivity/Impulsivity scale. Table 1 shows that the scores of children in the ADHD group were significantly higher compared to normal controls on the parent and teacher DBD scales of Inattention, Hyperactivity/Impulsivity, ODD and CD. To assess the level of chronic anxiety in children, all children completed the Dutch version of the Fear Survey Schedule for Children-Revised (FSSC-R; Ollendick, 1983). As shown by Table 1, no group differences were revealed on scores of total anxiety for the FSSC-R. Fourteen children with ADHD were treated with methylphenidate and discontinued use at least 36 hours prior to testing.

Normal control children were recruited from different local community schools. Control children with scores above the 80th percentile on *any* subscale of the parent or teacher DBD were excluded.

Time production task

Figure 1 shows the stimulus sequence in the time production task. Each trial commenced with a tone (80 db, 50 ms) after which children pressed a response button with their right index finger, when they thought a 1-second interval had elapsed. A 1500 ms delay screen separated the button press from the feedback information (500 ms). The inter-trial interval was 1500 ms. Accuracy feedback ('too short', 'too long' or 'correct') was provided using a staircase algorithm, which ensured similar levels of positive and negative feedback for each subject in each condition. The initial criterion for a response to be correct was a response between 500 and 1500 ms. Positive feedback was provided when response latencies fell within the boundaries of the current time window. After positive feedback, the boundaries narrowed by 50 ms on both sides of the window. After negative feedback ('too short' or 'too long'), the boundaries widened by 50 ms. Children were required to produce a 1-second interval under three reinforcement conditions: (1) feedback-only, (2) response cost: feedback and a 3 cents loss when responses were incorrect and (3) reward: feedback and 3 cents gain when responses were correct. The conditions were presented in a pseudo-random order. Loss or gain ('+3 cent', '–3 cent') were presented on the screen.

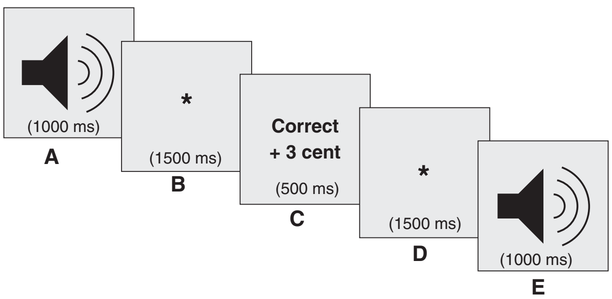

Figure 1 The stimulus sequence in the time production task (this example represents a reward-trial). A) Children heard an auditory beep (50 ms) indicating the start of a 1-second interval, while looking at a fixation cross on the screen. Approximately 1000 ms later, children pressed the response button; B) 1500 ms after pressing the button, C) feedback (correct, too short, too long) appeared on the screen for 500 ms. D) The screen goes blank for 1500 ms prior to the next trial

Stimuli were presented on a 17-inch computer screen, positioned 2.40 metres in front of the participant. To familiarise children with the 1-second interval, a cartoon character appeared on the screen 10 times for exactly 1 second. Children practised the task in a 30-trial practice session. Standardised task instructions were provided by the experimenter and the task commenced. Each condition consisted of 160 trials divided into two blocks. In the feedback-only condition, children were told to perform as accurately as possible. In the reward condition, similar instructions were provided and children were told to gain as much money as possible, which was put into a savings box in front of the children after each block. To make the task realistic, all children gained 80 cents after the first block and another 100 cents following the second block. In the response cost condition children received 350 cents at the start and they were told to lose as little as possible. From this, children lost 100 cents following the first block and 80 cents following the second block. The total gain after three reinforcement conditions was thus 350 cents. Heart rate was measured during the task.

ECG measurements

ECG was registered with two Ag/AgCl electrodes, attached between the collarbones over the jugular notch of the sternum and at the right lateral side between the lower two ribs. The continuous signals were amplified and sampled at 500 Hz. R-peak occurrences were detected by using peak detection software and stored offline. To include all validly recorded inter-beat intervals (IBIs) and exclude noise in the heart rate data, IBIs that were larger than 1500 ms and smaller than 400 ms were excluded. IBIs were extracted around the feedback moment. The interval in which the feedback took place was IBI0. IBI+1, IBI+2 and IBI+3 followed feedback, and the intervals preceding feedback were IBI–1, IBI–2, and IBI–3. Time production trials over 3 seconds and under 200 ms were excluded as being non-informative for our heart beat data (e.g., due to omissions and premature responses). This was .7% of the trials across groups.

Heart rate response

In order to investigate HRRs to reinforcement feedback, we explored the change in interval between successive IBIs following feedback. The effect of feedback on heart rate is found to be the most robust between IBI0 to IBI+2 following feedback (approximately 1800 ms; Crone et al., 2005; Somsen et al., 2000). IBIs were compared to a baseline IBI prior to the start of the trial. The baseline was a single value that was subtracted from the IBIs of interest in each individual trial. There were no main or interaction effects of group, condition or feedback that could influence the results for this IBI. The IBIs were submitted to an ANOVA with condition (feedback-only, response cost, reward), feedback (positive, negative) and sequential IBI (IBI0, IBI+1, IBI+2) as repeated measures and group (ADHD, normal controls) as between-subject factor.

Heart rate variability

IBI files were converted from the event domain (the IBI values) to the time domain (evenly spaced time points). HRV was measured using Fast Fourier Transform as calculated using a custom software program. The area under the curve for each subject was calculated for the low (.04–.08 Hz) and middle (.09–.15 Hz) frequency bands in the power spectrum, since these frequency bands are most sensitive to manipulations of task engagement (see Hyde & Izard, 1997). Concerning both blocks of the two reinforcement conditions, the last 340 seconds of data were analysed (represented as a time series of 512 data points sampled every 664 ms) to minimise practice effects. The spectral power index was expressed in deviations relative to the mean value of the time series of each block (squared modulation index). The time series were detrended with sequential cubic spines (see Hyde & Izard, 1997) to remove low frequency noise (e.g., movement artefacts).

In order to investigate task engagement in the reinforcement conditions, HRV was compared in an ANOVA with condition (feedback-only, response cost, reward) as repeated measures and group (ADHD, controls) as between-subject factor. When sphericity assumptions were violated, Greenhousse Geisser adjusted F-values and *p*-values are reported in parentheses for all dependent variables.

Results

Heart rate response to reinforcement feedback

Figure 2 shows baseline-corrected HRRs for children with ADHD and controls in the reinforcement conditions. Note that heart rate decreased between IBI–3 to IBI–1. This is a well-known phenomenon associated with preparatory motor responses (e.g., Jennings, Van der Molen, Brock, & Somsen, 1991) and is followed by heart rate recovery.

The planned analysis for IBI0 to IBI+2 following feedback revealed no significant main effect of group or condition (p values > .05). Negative feedback resulted in a lower heart rate compared to positive feedback, $F_{1,34} = 16.3$, $p = .000$, $\eta_p^2 = .32$. This effect did not differ between groups ($p > .05$). There was a main effect of sequential IBI, $F_{2,33} = 27.8$, $p = .000$, $\eta_p^2 = .63$, and an interaction between sequential IBI and group, $F_{2,33} = 3.3$, $p = .049$, $\eta_p^2 = .17$. Post-hoc analyses showed that controls exhibited a delay in heart rate recovery between IBI0 and IBI+1 (no difference, $p > .05$), while their heart rate accelerated between IBI+1 (29.4 ms) and IBI+2

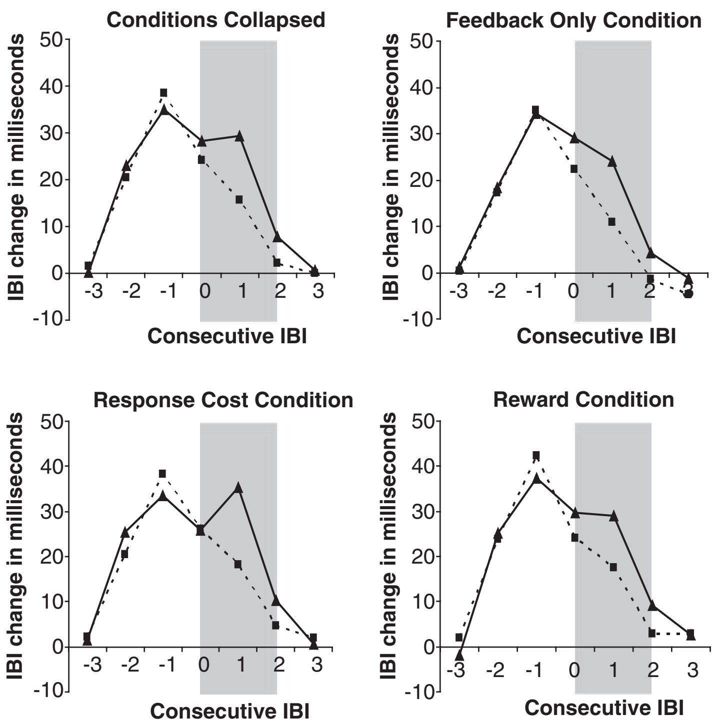

Figure 2 Heart rate responses following feedback for IBI0 to IBI+2 (gray area) collapsed across reinforcement conditions (2a) and in the reinforcement conditions (2b, c, d) of the time production task for children with ADHD (dotted lines) and controls (solid lines). Positive and negative feedback are collapsed. IBI0 indicates the interval in which the feedback was presented. IBI = Inter-Beat-Interval

(7.9 ms), $F_{1,17} = 53.3$, $p = .000$, $\eta_p^2 = .76$. In contrast, children with ADHD showed a linear increase in heart rate from IBI0 (24.1 ms) to IBI+1 (15.6 ms), $F_{1,17} = 6.6$, $p = .030$, $\eta_p^2 = .25$, to IBI+2 (2.1 ms), $F_{1,17} = 19.3$, $p = .000$, $\eta_p^2 = .53$. Other higher-order interactions involving group were not significant (*p*values > .05). Consequently, children with ADHD exhibited smaller HRRs than controls, irrespective of the reinforcement condition.

Heart rate variability

The effects of group, condition and the interaction between group and condition were not significant for HRV in the middle frequency band (*p* values > .05). In the low frequency band, group interacted with condition (see Figure 3), $F_{2,33} = 4.3$, $p = .015$, $\eta_p^2 = .22$ (Greenhouse Geisser $F_{2,33} = 3.5$, $p = .045$, $\eta_p^2 = .09$). Post-hoc analyses revealed that controls exhibited no difference in HRV between conditions ($p > .05$). In contrast, children with ADHD showed HRV suppression in the response cost, $F_{1,17} = 17.3$, $p = .001$, $\eta_p^2 = .51$, and reward conditions, $F_{1,17} = 8.6$, $p = .009$, $\eta_p^2 = .34$, compared to the feedback-only condition. There were no main effects of group or condition ($p > .05$) for HRV in the low frequency band. Taken together, the results suggest that children with ADHD show a lack of task engagement, when performance is not tightly coupled with incentives.

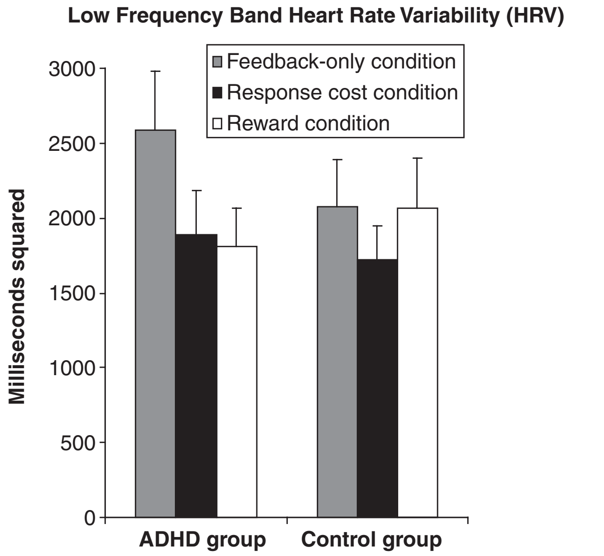

Figure 3 Task engagement in three reinforcement conditions of the time production task for children with ADHD and controls. Bars show the area under the curve (and standard errors) of power calculations in the low frequency band (.04–.08) of inter-beat-interval data. Lower values indicate less HRV and increased task engagement

Discussion

This is the first study that has employed HRRs and HRV to investigate the neurobiological processing of reinforcement feedback processing in ADHD. In a time production paradigm, participants received positive and negative feedback under three reinforcement conditions: feedback-only, response cost and reward. We tested (a) whether children with ADHD show disturbed reinforcement feedback processing by measuring HRR to reinforcement feedback, and (b) whether children with ADHD show motivational deficits (lack in task engagement) as measured by HRV. Our findings indicate that ADHD children have problems in monitoring feedback. Second, ADHD participants have poor task engagement when performance is not reinforced.

Reinforcement feedback processing

In both children with ADHD and controls, heart rate decelerated in preparation to making a response, and was followed by heart rate recovery after the response. ADHD children showed smaller HRRs to feedback compared to controls, as demonstrated by the interaction between group and sequential IBI. Controls initially showed delayed heart rate recovery following feedback (IBI0 to IBI+1), after which heart rate accelerated (IBI+1 to IBI+2). ADHD children, in contrast, showed immediate heart recovery following the button press in all reinforcement conditions. From the literature, there are several interpretations on impaired HRRs to feedback which are related to problems with: (a) feedback processing with the purpose of adjusting future performance (Crone et al., 2005), (b) motivational evaluation of feedback (Van der Veen et al., 2004), and (c) performance monitoring and feedback expectancy (Somsen et al., 2000). Performance data of our study indicated that timing adjustment on the trials following negative feedback ('too short' or 'too long') did not differ between children with ADHD and controls ($p > .05$), which argues against the first hypothesis (a). Processing of the motivation-related features of feedback (hypothesis b) seems intact in children with ADHD, since the discrimination between positive and negative feedback did not differ between groups and there was no condition by group interaction. Therefore, the interpretation that feedback expectancy is impaired in children with ADHD seems most plausible (hypothesis c). HRRs to feedback are suggested to be larger when the difference between the 'expected' and 'actual' feedback is larger (Somsen et al., 2000) and due the dynamic tracking algorithm, feedback expectancy is not dependent on performance monitoring only. In control children, 50% positive feedback may have been worse than expected in terms of performance, which resulted in a sharpened HRR to feedback. Children with ADHD, due to a learned history of blunted performance, demonstrated abnormal performance expectancies which resulted in smaller HRRs to feedback as compared to controls.

An unexpected event may trigger an attention response (Jennings et al., 1991), and impaired allocation of attention following feedback in children with ADHD may explain the findings of response timing problems in ADHD in the current study.

Task engagement

The increased low frequency HRV (ARV band) in the absence of reinforcement contingencies suggests a lack of task engagement in children with ADHD when reinforcement is not available. This is in line with the suggestion that children with ADHD suffer from a motivation deficit and cannot keep up with task demands due to a non-optimal energetic state (Sergeant et al., 1999). They dependent on external motivators such as reward and response cost in order to increase their motivation and perform well. Lower ARV rhythmicity is associated with a diminished bloodflow that regulates the local cerebral metabolic demands, for example, during task performance (Hyde & Izard, 1997). Whereas two previous studies showed group differences in HRV between ADHD children and controls during a cognitive task (Börger et al., 1999; Börger & Van der Meere, 2000), the current findings demonstrate that these differences can be modified by reinforcement contingencies. The findings are in line with suggestions that the pathogenesis of ADHD should be conceptualised from a perspective other than a purely cognitive one (Castellanos & Tannock, 2002; Sonuga-Barke, 2002; Sergeant et al., 1999). Sonuga-Barke (2002) proposed two dysfunctional brain pathways that reciprocally interact in explaining the pathogenesis of ADHD. The pathways are responsible for cognitive deficits on the one hand and motivational deficits on the other. The motivation deficits are explained by a shortage in extracellular dopamine in the striatum that reduces the impact of reinforcement on behaviour (Sagvolden et al., 2005).

An alternative theoretical focus is to classify increased HRV as being part of variability in several domains of functioning. Besides greater variability in heart rate (Börger et al., 1999), children with ADHD are characterised by an enhanced level of symptom variability (Castellanos & Tannock, 2002) and behavioural variability: more variable response times (Leth-Steensen, Elbaz, & Douglas, 2000), time estimation (e.g., Van Meel et al., 2005b), and increased variability in the P300 following feedback (Lazzaro et al., 1997). Castellanos et al. (2005) speculated that children with ADHD show a deficiency in reducing low frequency fluctuations in neuronal activity that causes lapses of attention and which is responsible for attention problems, forgetfulness, and unexpected errors. There is evidence of decreased response variability in ADHD when performance is reinforced (Van Meel et al., 2005b); however, future studies need to investigate whether the impact of motivation on response variability and psychophysiological variability are associated.

Heart rate and performance

Behavioural data from this study (Van Meel et al., 2005b) showed that children with ADHD improved their performance under conditions of reinforcement. However, despite normal psychophysiological task engagement under reinforcement, children with ADHD were less accurate in time production than controls. This suggests, firstly, that ADHD children may need to compensate for deficits other than solely a motivational dysfunction (in this case timing dysfunctions). Compensation mechanisms in ADHD have been reported in the neuro-imaging literature (Bush et al., 1999; Johnstone & Barry, 1996), whereby children with ADHD showed increased brain activation in other areas than controls (Fassbender & Schweitzer, 2006). A second possibility is that the reinforcers were not sufficiently salient for children with ADHD. Children with ADHD may need larger reinforcement in order to perform as well as controls (Slusarek et al., 2001). A third possibility is that neurobiological sensitivity may differ between children with ADHD and controls (Fassbender & Schweitzer, 2006), for example due to deviation in brain development. This suggests that the psychophysiological activity that correlates with behaviour may differ between children with ADHD and controls.

Heart rate variables in our study suggest that children with ADHD suffer from difficulties in motivating themselves and performing well when reinforcement is not available. If children with ADHD show impaired feedback monitoring as compared to controls, this may have triggered less 'attention' to the task. Children with ADHD may need reinforcement in addition to feedback for (a) more efficient shaping of behaviour, for example by controlling their responses and hence becoming less variable, or (b) an increase in the subjective value of feedback in order to boost their motivation.

Limitations

Firstly, the small sample size demands verification of our findings in a larger sample. Secondly, children with ADHD had lower IQ scores than controls. Kuntsi et al. (2004) demonstrated that correlations between ADHD and IQ are due to a genetic component. The relation between IQ and HRR or HRV is unknown; in our study we found no meaningful correlations between IQ and the dependent variables (all p values > .05), suggesting no confounding effects of IQ. The third issue is the presence of oppositional and aggressive behaviour in the ADHD sample, as indicated by scores on the DISC and DBD. There is substantial overlap between ADHD and ODD/CD, and with ADHD and anxiety disorders (Angold, Costello, & Erkanli, 1999). Studies have found diminished sensitivity to punishment in ODD/CD groups as measured by both behavioural and psychophysiological recordings (Herpertz et al., 2001; Newman, Wallace, Schmitt, & Arnett, 1997). There are indications of increased heart rate responses to feedback in anxious individuals (Crone et al., 2005). We examined the potential contribution of DBD parent- and teacher-reported ODD and CD symptoms and the self-report measure of anxiety to our dependent variables, and no meaningful correlations were revealed (all *p* values > .05).

Conclusion

The present study demonstrated that heart rate variables can provide insight into processes that are not easily detectable using performance measures: ADHD is characterised by impaired monitoring of feedback and reduced task engagement. Motivational deficits at a psychophysiological level have been identified earlier in ADHD using fMRI (Scheres, Milham, Knutson, & Castellanos, 2005). If replicated, the current findings open up a window for research into the practical consequences of reinforcement feedback in ADHD. Issues to be studied include whether possible abnormal motivation and impaired monitoring of feedback affects academic performance in children with ADHD, whether the enhanced impact of reinforcement using pharmacological treatment influences performance, and whether such findings influence the psychosocial management of children with ADHD. From a clinical perspective, feedback is a crucial concept in behavioural therapy (Milne & James, 2000). Hence, analysis of feedback and its biological underpinning can potentially assist in developing therapeutic procedures. Interventions for ADHD need to be designed that take account of diminished monitoring of feedback and lack of task engagement by, for example, making feedback more salient or systematically changing the subjective value of reinforcement.

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References

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

Börger, N., Van der Meere, J.J., Ronner, A., Alberts, E., Geuze, R., & Bogte, H. (1999). Heart rate variability and sustained attention in ADHD children. *Journal of Abnormal Child Psychology*, *27*, 25–33.

Börger, N., & Van der Meere, J.J. (2000). Motor control and state regulation in children with ADHD: A cardiac response study. *Biological Psychology*, *51*, 247–267.

Bush, G., Frazier, J.A., Rauch, S.L., Seidman, L.J., Whalen, P.J., Jenike, M.A., Rosen, B.R., & Biederman, J. (1999). Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder revealed by fMRI and the Counting Stroop. *Biological Psychiatry*, *45*, 1542–1552.

Cameron, J., & Pierce, W.D. (1994). Reinforcement, reward, and intrinsic motivation: A meta-analysis. *Review of Educational Research*, *64*, 363–423.

Carlson, C.L., Booth, J.E., Shin, M.S., & Canu, W.H. (2002). Parent-, teacher-, and self-rated motivational styles in ADHD subtypes. *Journal of Learning Disabilities*, *35*, 104–113.

Castellanos, F.X., Sonuga-Barke, E.J., Scheres, A., Di Martino, A., Hyde, C., & Walters, J.R. (2005). Varieties of attention-deficit/hyperactivity disorder-related intra-individual variability. *Biological Psychiatry*, *57*, 1416–1423.

Castellanos, F.X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: The search for endophenotypes. *Nature Reviews Neuroscience*, *3*, 617–628.

Crone, E.A., Bunge, S.A., De Klerk, P., & Van der Molen, M.W. (2005). Cardiac concomitants of performance and individual monitoring: Context dependence and individual differences. *Cognitive Brain Research*, *23*, 93–106.

Crone, E.A., Jennings, J.R., & Van der Molen, M.W. (2003). Sensitivity to interference and response contingencies in attention-deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry*, *44*, 214–226.

Douglas, V.I. (1989). Can Skinnerian theory explain attention deficit disorder. A reply to Barkley. In L.M. Bloomingdale & J.A. Sergeant (Eds.), *Attention deficit disorder: Current concepts and emerging trends in attentional and behavioral disorders of childhood* (pp. 235–54). Elmsford, NY: Pergamon.

Doyle, A.E., Faraone, S.V., Seidman, L.J., Willcutt, E.G., Nigg, J.T., Waldman, I.D., Pennington, B.F., Peart, J., & Biederman, J. (2005). Are endophenotypes based on measures of executive functions useful for molecular genetic studies of ADHD? *Journal of Child Psychology and Psychiatry*, *46*, 774–803.

Fassbender, C., & Schweitzer, J.B. (2006). Is there evidence for neural compensation in attention deficit disorder? A review of the functional neuroimaging literature. *Clinical Psychology Review*, *26*, 445–465.

Groth-Marnat, G. (1997). *Handbook of psychological assessment* (3rd edn). New York: Wiley.

Herpertz, S.C., Wenning, B., Mueller, B., Qunaibi, M., Sass, H., & Herpertz-Dahlmann, B. (2001). Psychophysiological responses in ADHD boys with and without conduct disorder: Implications for adult antisocial behavior. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 1222–1230.

Hyde, C.T., & Izard, C.E. (1997). Cardiac rhythmicities and sustained attention in children. *Psychophysiology*, *34*, 547–552.

Iaboni, F., Douglas, V.I., & Ditto, B. (1997). Psychophysiological response of AD/HD children to reward and extinction. *Psychophysiology*, *34*, 116–23.

Jennings, J.R., Van der Molen, M.W., Brock, K., & Somsen, R.J.M. (1991). Response inhibition initiates cardiac deceleration: Evidence from a sensory-motor compatibility paradigm. *Psychophysiology*, *28*, 72–85.

Johnstone, S.J., & Barry, R.J. (1996). Auditory event-related potentials to a two-tone discrimination paradigm in attention deficit hyperactivity disorder. *Psychiatry Research*, *64*, 179–92.

Jorna, P.G.A.M. (1992). Spectral-analysis of heart-rate and psychological state – a review of its validity as a workload index. *Biological Psychology*, *34*, 237–57.

Keitz, M., Martin-Soelch, C., & Leenders, K.L. (2003). Reward processing in the brain: A prerequisite for movement preparation? *Neural Plasticity*, *10*, 121–128.

Kuntsi, J., Eley, T.C., Taylor, A., Hughes, C., Asherson, P., Caspi, A., & Moffitt, T.E. (2004). Co-occurrence of ADHD and low IQ has genetic origins. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, *124*, 41–47.

Lazzaro, I., Anderson, J., Gordon, E., Clarke, S., Leong, J., & Meares, R. (1997). Single trial variability within the P300 (250–500 ms) processing window in adolescents with attention deficit hyperactivity disorder. *Psychiatry Research*, *73*, 91–101.

Leth-Steensen, C., Elbaz, Z.K., & Douglas, V.I. (2000). Mean response times, variability, and skew in the responding of ADHD children: A response time distributional approach. *Acta Psychologica*, *104*, 167–90.

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*, 377–88.

Luman, M., Oosterlaan, J., & Sergeant, J.A. (2005). The impact of reinforcement contingencies on AD/HD: A review and theoretical appraisal. *Clinical Psychology Review*, *25*, 183–213.

Milne, D., & James, I. (2000). A systematic review of effective cognitive-behavioural supervision. *British Journal of Clinical Psychology*, *39*, 111–127.

Mulder, G., & Mulder, L.J.M. (1981). Information processing and cardiovascular control. *Psychophysiology*, *18*, 302–402.

Newman, J.P., Wallace, J.F., Schmitt, W.A., & Arnett, P.A. (1997). Behavioral inhibition system functioning in anxious, impulsive and psychopathic individuals. *Personality and Individual Differences*, *23*, 583–592.

Ollendick, T.H. (1983). Reliability and validity of the Revised Fear Survey Schedule for Children (FSSC-R). *Behavioral Research and Therapy*, *21*, 685–692.

Pelham, W.E., Gnagy, E.M., Greenslade, K.E., & Milich, R. (1992). Teacher ratings of DSM-III-R symptoms for the disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 210–218.

Porges, S.W. (1991). Vagal tone: An autonomic mediator of affect. In J. Garber & K.A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp. 111–128). Cambridge, UK: Cambridge University Press.

Pruyn, A., Aasman, J., & Wyers, B. (1985). Social influences on mental processes and cardiovascular activity. In J.F. Orlebeke, G. Mulder & L.J.P. Van Doornen (Eds.), *The psychophysiology of cardiovascular control (models, methods, and data)* (pp. 865–877). New York: Plenum Press.

Sagvolden, T., Johansen, E.B., Aase, H., & Russell, V.A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences*, *28*, 397–419.

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.

Scheres, A., Milham, M.P., Knutson, B., & Castellanos, F.X. (in press). Ventral striatal hyporesponsiveness during reward anticipation in attention-deficit/hyperactivity disorder. *Biological Psychiatry*.

Schultz, W. (2000). Multiple reward signals in the brain. *Nature Reviews Neuroscience*, *1*, 199–207.

Sergeant, J.A., Oosterlaan, J., & Van der Meere, J.J. (1999). Information processing in attention-deficit/hyperactivity disorder. In H.C. Quay & A.E. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 75–104). New York: Plenum Press.

Sergeant, J.A., & Van der Meere, J.J. (1988). What happens after a hyperactive child commits an error? *Psychiatry Research*, *24*, 157–64.

Shaffer, D., Fisher, P., Lucas, C.P., Dulcan, M.K., & Schwab-Stone, M.E. (2000). NIMH Diagnostic Interview Schedule for Children version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 28–38.

Slusarek, M., Velling, S., Bunk, D., & Eggers, C. (2001). Motivational effects on inhibitory control in children with AD/HD. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 355–363.

Somsen, R.J., Van der Molen, M.W., Jennings, J.R., & Van Beek, B. (2000). Wisconsin Card Sorting in adolescents: Analysis of performance, response times and heart rate. *Acta Psychologica*, *104*, 227–257.

Sonuga-Barke, E.J. (2002). Psychological heterogeneity in AD/HD – a dual pathway model of behaviour and cognition. *Behavioural Brain Research*, *10*, 29–36.

Suess, P.E., Newlin, D.B., & Porges, S.W. (1997). Motivation, sustained attention, and autonomic regulation in school-age boys exposed in utero to opiates and alcohol. *Experimental and Clinical Psychopharmacology*, *5*, 375–387.

Van der Veen, F.M., Van der Molen, M.W., Crone, E.A., & Jennings, J.R. (2004). Phasic heart rate responses to performance feedback in a time production task: Effects of information versus valence. *Biological Psychology*, *65*, 147–161.

Van Meel, C.S., Oosterlaan, J., Heslenfeld, D.J., & Sergeant, J.A. (2005a). Telling good from bad news: ADHD differentially affects processing of positive and negative feedback during guessing. *Neuropsychologia*, *43*, 1946–1954.

Van Meel, C.S., Oosterlaan, J., Heslenfeld, D.J., & Sergeant, J.A. (2005b). Motivational effects on motor timing in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 451–460.

Volkow, N.D., Wang, G.J., Fowler, J.S., Telang, F., Maynard, L., Logan, J., Gatley, S.J., Pappas, N., Wong, C., Vaska, P., Zhu, W., & Swanson, J.M. (2004). Evidence that methylphenidate enhances the saliency of a mathematical task by increasing dopamine in the human brain. *American Journal of Psychiatry*, *161*, 1173–1180.

Wiersema, J.R., Van der Meere, J., & Roeyers, H. (2005). ERP correlates of impaired error monitoring in children with ADHD. *Journal of Neural Transmission*, *112*, 1417–1430.

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