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Abstract: Children with attention deficit/hyperactivity disorder (ADHD) choose smaller sooner (SS) over larger later (LL) rewards more than controls. Here we assess the contributions of impulsive drive for immediate rewards (IDIR) and delay aversion (DAv) to this pattern. We also explore the characteristics of, and the degree of familiality in, ADHD SS responders. We had 360 ADHD probands; 349 siblings and 112 controls (aged between 6 to 17 years) chose between SS (1 point after 2 s) and LL reward (2 points after 30 s) outcomes on the Maudsley Index of Delay Aversion (Kuntsi, Oosterlaan, Stevenson, 2001): Under one condition SS choice led to less overall trial delay under another it did not. ADHD participants chose SS more than controls under both conditions. This effect was larger when SS choice reduced trial delay. ADHD SS responders were younger, had lower IQ, more conduct disorder and had siblings who were more likely to be SS responders themselves. The results support a dual component model in which both IDIR and DAv contribute to SS choice in ADHD. SS choice may be a marker of an ADHD motivational subtype. (PsycINFO Database Record (c) 2009 APA, all rights reserved).

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Delay and Reward Choice in ADHD: An Experimental Test of the Role of Delay Aversion

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Children with attention deficit/hyperactivity disorder (ADHD) choose smaller sooner (SS) over larger later (LL) rewards more than controls. Here we assess the contributions of impulsive drive for immediate rewards (IDIR) and delay aversion (DAv) to this pattern. We also explore the characteristics of, and the degree of familiality in, ADHD SS responders. We had 360 ADHD probands; 349 siblings and 112 controls (aged between 6 to 17 years) chose between SS (1 point after 2 s) and LL reward (2 points after 30 s) outcomes on the Maudsley Index of Delay Aversion (Kuntsi, Oosterlaan, & Stevenson, 2001): Under one condition SS choice led to less overall trial delay under another it did not. ADHD participants chose SS more than controls under both conditions. This effect was larger when SS choice reduced trial delay. ADHD SS responders were younger, had lower IQ, more conduct disorder and had siblings who were more likely to be SS responders themselves. The results support a dual component model in which both IDIR and DAv contribute to SS choice in ADHD. SS choice may be a marker of an ADHD motivational subtype.

Keywords: attention deficit/hyperactivity disorder, delay aversion, children, adolescents

Although neuropsychological models of attention deficit/hyperactivity disorder (ADHD) have often focused on the role of cognitive deficits, motivational factors have also been implicated in the disorder (Castellanos, Sonuga-Barke, Tannock, & Milham, 2006). A recent review concluded that, on the basis of existing data, one of the most robust motivational markers in ADHD was a preference for smaller sooner (SS) over larger later (LL) rewards (Luman, Oosterlaan, & Sergeant, 2005). This pattern of choice has been shown in most (e.g., Antrop et al., 2006; Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009; Dalen, Sonuga-Barke, & Remington, 2004; Hoerger & Mace, 2006; Kuntsi, Oosterlaan, & Stevenson, 2001; Schweitzer & Sulzer-Azaroff, 1995; Solanto et al., 2001; Sonuga-Barke, Taylor, Sembi, & Smith, 1992), but not all (Bidwell, Willcutt, DeFries, & Pennington, 2007; Scheres et al., 2006; Solanto et al., 2007) studies of ADHD using SS versus LL choice paradigms. A recent meta-analysis reported a pooled effect size for case-control differences of a similar magnitude to those seen for executive function measures (Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008).

This effect has been explained in a number of ways. Deficit in inhibitory control, part of a broader pattern of executive dysfunction (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2008), may mean that children with ADHD find it difficult to withhold their response from the SS option even when the LL one is more valuable. Alternatively, they may have a steepened delay of reinforcement gradient (Sagvolden, Johansen, Aase, & Russell, 2005; Tripp & Wickens, 2008) leading to sharper discounting of the value of LL (Barkley et al., 2001). These two possibilities, although derived from different theoretical perspectives, share one important common element: The key intertemporal determinant of SS over LL choice is hypothesized to be the relative delay before the LL and the SS options so that increases in LL prereward delay (preRD) increase the preference of SS over LL, all else remaining equal. This choice pattern is often regarded as an index of impulsiveness (Schweitzer & Sulzer-Azaroff, 1995). The somewhat more specific term *impulsive drive for immediate reward* (IDIR) will be employed in this paper, to distinguish SS over LL choice from the broader clinical connotation of the term impulsiveness.

The concept of delay aversion (DAv) offers an alternative to IDIR as an explanation of SS preference in ADHD (Sonuga-Barke et al., 1992). According to this account, choice is driven not by an impulsive drive for immediate reward, but rather by a generalized aversion to delay. This aversiveness is hypothesized to derive from the fact that delay has an especially strong negative affective valence for children with ADHD. This is manifest in feelings of frustration, agitation, and emotional arousal when delay is imposed. Consistent with this view, children with ADHD display patterns of vigilance to delay-related cues similar to those shown by anxious children faced with physical and social threat (Sonuga-Barke, De Houwer, De Ruiter, Azensten, & Holland, 2004). They also display heightened levels of frustration during long and boring tasks (Bitsakou et al., 2009; Scime & Norvilitis, 2006). According to the DAv model, the primary motivating factor for ADHD SS choice is the escape or avoidance of delay before LL, (rather than access to the SS reward), and the reduction in negative affect that this achieves. The child with ADHD's SS preference is therefore maintained by a process of negative rather than positive reinforcement. Furthermore, in contrast to IDIR models delay both before and after the delivery of rewards (as components of total delay [TD]) are predicted to be equally influential in determining of children with ADHD's choice of SS over LL. Crucially, the consequences of this generalized pattern of DAv are predicted to be seen in a broader pattern of delay-related effects on functioning (Sonuga-Barke, 1994, 2003); even in situations in which TD cannot actually be reduced (i.e., fixed-delay nonchoice situations). In these situations DAv is seen in patterns of delay-induced inattention and hyperactivity that in turn can lead to performance deficits in such settings. Consistent with this, children with ADHD show increased activity and responding during fixed periods of

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delay or extinction of reinforcers (Antrop, Roeyers, Van Oost, & Buysse, 2003; Sagvolden, Johansen, Aase, & Russell, 1998). They also disengage from long and boring tasks with the passage of time as their attention to nontask related activity increases. Their performance is differentially affected by slow event rates (Aase & Sagvolden, 2006; Wiersema, van der Meere, Roeyers, Van Coster, & Baeyens, 2006). According to the model this broader pattern of delay-related effects is maintained because it reduces the perceived duration of delay (i.e., makes the delay pass more quickly) even when actual delay cannot be altered (Sonuga-Barke, 1994).

What is the current evidence that DAv contributes to the SS preference in ADHD? Unfortunately, despite the obvious importance of identifying its causes, previous research often has not been able to determine the relative contributions of DAv and IDIR to SS preference in ADHD because of limitations in experimental design. Studies typically have used a single delay condition characterized by the repeated presentation of choices between SS and LL; with the delivery of rewards after delay being followed immediately by the end of one trial and the start of the next. This design feature means that choosing the SS reduces both preRD and TD simultaneously (e.g., Bitsakou et al., 2009; Solanto et al., 2001). This means that the effects of SS choices on preRD and TD are completely confounded and their relative contributions cannot be distinguished. Some studies have addressed this confound by employing a fixed-length trial format in which choosing the SS reduces preRD but does not reduce TD. This has been achieved by adding a period of postreward delay (postRD) after the delivery of rewards (SS or LL, respectively) that is the same length of the prereward delay period of the other option (LL or SS, respectively; Schweitzer & Sulzer-Azaroff, 1995). Under this condition, SS choices do not reduce TD so that ADHD-related SS preference is deemed to be due to IDIR. However, this condition does not allow an assessment of the contribution of DAv over and above the effects of IDIR.

Such an assessment can only be achieved by contrasting SS choices under the two conditions in which choices between SS and LL are presented with or without postRD (Figure 1a). For such a comparison the following key predictions are made. First, if ADHD children choose the SS more than controls on both the postRD and no-postRD conditions, and the size of the case-control difference is the same under both conditions (i.e., removing the postRD periods does not increase preference for SS) then the role of IDIR is supported and the role of DAv is refuted. Second, if they choose SS more only under the no-postRD condition then the role for DAv is supported and that for IDIR refuted. Third, if SS is chosen more by children with ADHD under both conditions—but the effect is significantly larger in the no-postRD condition than in the postRD condition (i.e., linking the choice of the SS to a reduction in TD increases the effect) then a dual component determination of SS preference in ADHD is supported in which the drive to escape delay associated with DAv compounds the impulsive drive for immediate reward seen on the postRD condition and exacerbates the preference for SS over LL in the no-postRD delay condition. The three different predictions are illustrated in Figure 1b.

To date there have been no studies published comparing clinically diagnosed ADHD and unaffected control children's SS choices under these two conditions. This means that the relative contribution of IDIR and DAv to ADHD SS choice is unknown. Two studies have reported a comparison of more or less inattentive/overactive children (one with school aged and one preschool children) in which assignment of participants was based on subclinical cut-offs on a dimensional measure of symptoms and children were drawn from a normal population (Dalen et al., 2004; Sonuga-Barke et al., 1992). The results from these two studies were similar. Both found that when there was a limited number of choices to make both high and low inattention/overactive groups favored the LL under the postRD condition, whereas under the no-postRD condition the high inattention/overactive group, but not the low group, favored the SS. Against expectation the results therefore supported the DAv model over the IDIR and the two component models. Children with high hyperactivity only chose the SS when this reduced TD. These findings were particularly surprising as they seemed to run counter to the commonly accepted idea that children hyperactivity were impulsive in the sense that they find it difficult to wait for LL (Sonuga-Barke, 1994).

Because of the failure to test and therefore to replicate these experimental effects in a clinical sample of diagnosed ADHD cases the possibility remains that their relevance is limited to less severe and subclinical expressions and that the results would not generalize to a group of more impaired clinical cases. The primary aim of the current paper therefore is to test the predictions of the IDIR, DAv, and dual component models using a simple SS versus LL choice task (The Maudsley Index of Delay Aversion; MIDA; Kuntsi et al., 2001) with both postRD and no-postRD conditions in a large sample of diagnosed ADHD cases. There is evidence in the literature to support the view that IDIR is stronger for diagnosed cases than was seen in the Sonuga-Barke et al. (1992) and Dalen et al. (2004) studies with nonclinical cases. For instance, studies using diagnosed cases have shown an effect of reward immediacy even in situations incorporating fixed delay (Schweitzer & Sulzer-Azaroff, 1995; Tripp & Alsop, 2001). The current literature therefore favors a dual component model of ADHD SS preference. This leads to the prediction that children with ADHD will prefer SS more than controls under both postRD and no-postRD conditions but that the preference would be stronger under the no-postRD condition in which escape from delay exacerbates the IDIR seen under the postRD condition. In statistical terms we would therefore predict a main effect of group with ADHD choosing SS more than controls overall and an interaction between condition and group.

Models of ADHD have, in the last few years, begun to emphasize the heterogeneity of the disorder and the existence of multiple-putative causal pathways, mediated by different neuropsychological processes (Nigg, 2006; Pennington, 2006; Sergeant, Willcutt, & Nigg, 2008; Sonuga-Barke, 2002; Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). This has led to the formulation of the hypothesis that there are a number of, more or less, distinguishable subgroups of individuals with ADHD, each characterized by a particular profile of neuropsychological impairment (Sonuga-Barke, Sergeant, et al., 2008). A preliminary case for a "cognitive" ADHD subgroup marked by executive dysfunction (Nigg, 2005) has been made (Biederman et al., 2007; Nigg, Willcut, Doyle, & Sonuga-Barke, 2005), Nigg et al. (2005) demonstrated that only a minority of children with ADHD have deficits expressed to an abnormal degree of severity; a finding in keeping with the moderate case-control effect sizes found for most executive function test measures (Sergeant et al., 2008). The question of whether subgroupings of this kind are of clinical significance (in terms of

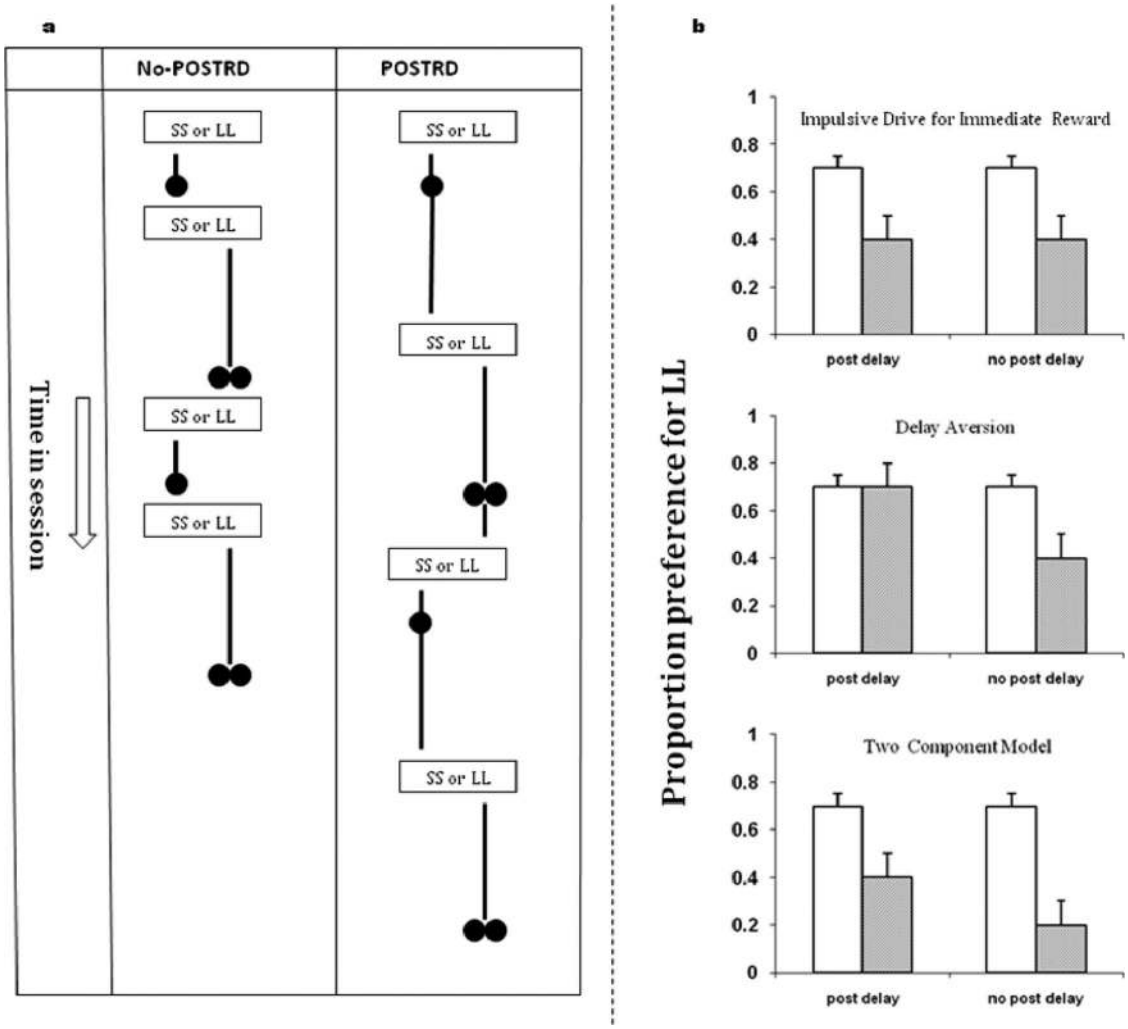

Figure 1. Figure 1a illustrates the delay (post and pre) and reward parameters of postRD and no-postRD conditions. The filled circles represent reward and the lines the length of delay. In the no-postRD condition choosing the SS reduces overall delay in the postRD condition it does not. Figure 1b illustrates the three predictions for the effects of postRD and no-postRD on SS choice as described in the text.

etiology, prognosis, and treatment response) remains to be addressed. A secondary and more exploratory aim of the current paper therefore is to determine to what extent a subgroup of children with ADHD, marked by a preference for SS, can be identified and to examine how they differ from other children with ADHD. This was possible because of the very large number of ADHD cases included in the study. We were especially interested in clinical presentation and comorbidities, background characteristics, and the extent to which SS preference is also displayed by children with ADHD's family members—in this case their siblings. A key question is: Do levels of SS choice cosegregate with ADHD within families? This question is related to the broader and substantive issue of whether family based etiological effects in ADHD (e.g., genes and shared family environments) are mediated by intervening neuropsychological processes (so called neuropsychological endophenotypes; Coghill, Nigg, Rothenberger, Sonuga-Barke, & Tannock, 2005). Most attempts to identify endophenotypes in ADHD have focused on executive and other cognitive functions (Doyle et al., 2005). However, recent studies suggest that performance variability, perhaps under the influence of motivational or energetic factors, could be important too (Andreou et al., 2007; Bidwell et al., 2007). Twin studies have reported low levels of heritability but significant effects of shared environment for SS over LL choice (Kuntsi et al., 2006; Kuntsi & Stevenson, 2001; but see Bidwell et al., 2007). However, these results may have been affected by the skewed distributions of choice data and could be the result of artificially inflated similarities between twin-pair members that limited the ability of models to provide accurate estimates of heritability.

In the current study to avoid this problem we adopted a different strategy. First we define group membership (SS versus LL responders) on the basis of children's choices made under the no-postRD condition. We focused on this condition on the grounds that this is where SS preference is predicted to be most pronounced

because of the conjunction of the influence of the two components IDIR and DAv. From a purely practical perspective this condition is predicted to give the largest range of performance and the highest level of SS preference. We define SS responders as those choosing the LL on 50% of trials or less and LL responders as those choosing it on more than 50% of trials. This threshold, although inevitably somewhat arbitrary, corresponds to approximately the 10th percentile of the score of the control participants group (8.9% of controls chose the LL in the no-postRD on 50% or more trials); a similar cut-off to the equivalent 90th percentile used by Nigg et al. (2005) in similar analyses. We compared SS and LL responders in terms of their characteristics, and those of their siblings to see if preference for SS over LL cosegregates in families.

Although primarily exploratory in nature, there are a number of predictions in relation to this second aim. First, that relative to LL responders we predicted that ADHD cases would have more oppositional conduct problems, but will not differ on the severity of ADHD symptoms. This prediction is derived from the idea that affect regulation may be an important component of the disorder for a reactive and aggressive subgroup of children with ADHD (Hinshaw, 2003), and that such a group might be marked by a more intense emotional response to external barriers to important outcomes such as those associated with the imposition of delay. An analysis of anxiety and depression was also included in an attempt to demonstrate the specificity of the conduct problem effect. Second, we predicted that SS and LL responders will not differ on factors such as IQ indicative of a cognitive deficit. Third, we predicted that they will have siblings who are more likely to be SS responders themselves; a finding that would be consistent with the idea of a familial element in the role of motivational deficits in ADHD. We were unable in the current analysis to establish whether familiality in SS choices was specific to ADHD cases as we could not explore the patterns of SS choice in the siblings of unaffected controls (see Method section).

The current study included children aged from 6 to 17 years and provides the first published analysis of the SS over LL choice in adolescents with ADHD. Symptoms of inattention become more prominent, and hyperactivity, less prominent as children move into adolescence (Feifel, 1996; Kaplan & Stevens, 2002). Furthermore, patterns of the motivational salience of outcomes undergo a qualitative change as people age across the life span with small rewards becoming far less salient as children grow into adolescence and access to rewards increases enormously (Bjork et al., 2004). This is particularly so with regard to monetary rewards, which are often used in choice studies with children (Wulfert, Block, Santa Ana, Rodriguez, & Colsman, 2002). At the same time, the ability to tolerate delay prior to the delivery of reward seems to develop very rapidly as children grow into adolescents (Green, Fry, & Myerson, 1994; Green, Myerson, Lichtman, Rosen, & Fry, 1996). The current study therefore will also provide the first evidence regarding the value of a simple choice paradigm pitting SS against LL choice for differentiating children with ADHD from healthy controls in the adolescent period.

Method

Participants

The clinical sample is a subset of the ADHD probands and their siblings included in the National Institutes of Mental Health (NIMH) funded International Multicenter ADHD Genetics (IMAGE) project (Andreou et al., 2007; Brookes et al., 2006; Chen et al., 2008; Lasky-Su et al., 2007; Sonuga-Barke, Brookes, et al., 2008b). Participants for the current project were recruited through specialist ADHD clinics at eight sites from seven countries from families of European/White descent with a clinically diagnosed *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., [*DSM–IV*], American Psychiatric Association, 1994) child with ADHD-combined type between 6 and 17 years of age, and with at least one sibling in the same age range. In addition to executing the general IMAGE protocol these sites also took part in a collaborative study to investigate the neuropsychological basis of ADHD (Ghent, Belgium; Dublin, Ireland; Tel Aviv, Israel; Goettingen, Essen, Germany; Valencia, Spain; Zurich, Switzerland, and London, England). The MIDA task was one of three neuropsychological tests that were administered at these sites.

Probands

Potential ADHD cases ($N = 376$) ascertained on the basis of rigorous clinical evaluation were additionally screened for ADHD symptoms using Conners's Rating Scales, Parent (CPRS–R; Conners, Sitarenios, Parker, & Epstein, 1998a) and Teacher (CTRS–R; Conners, Sitarenios, Parker, & Epstein, 1998b) versions. Those that met criteria (T score > 63), and had an IQ score of 70 or over, were administered a Parental Account of Children's Symptoms (PACS; Chen & Taylor, 2006); a clinical interview that operationalizes the *DSM–IV* criteria to give an ADHD diagnosis. Full MIDA data (i.e., from both conditions) was available for 360 probands between the ages of 6 and 17 with a clinical research diagnosis of ADHD. The majority met PACS diagnosis for combined type ($N = 346$). Four probands had a diagnosis of hyperactive/impulsive subtype and 10 had a diagnosis of predominantly inattentive type.

Siblings

There were 374 probands' siblings with ADHD available for the study. Full MIDA data was available from 349 of these. Siblings were also screened for ADHD and if they met the inclusion threshold a PACS interview was administered to confirm the diagnosis. Fifty-six siblings met the criteria for one of the ADHD subtypes (hyperactive/impulsive = 10; inattentive = 9; combined type = 37). Participants were excluded from the study if they showed evidence of a pervasive developmental disorder, neurological diseases, or other medical and genetic disorders that could mimic ADHD-like symptoms. As part of the general IMAGE study protocol, parents gave written consent for the children to participate in the study.

Healthy Controls

Three of the sites (Goettingen, Germany; London, England; and Valencia, Spain) participating in the study in addition recruited and administered the same cognitive tasks to a smaller sample of controls. These were recruited from mainstream primary and secondary school classes covering the same age as the probands. One site (London) collected data from control sibling pairs. For the purposes of the analyses included in the current paper, only one of the two control siblings was included (the one matched for age and sex with the proband). Although this sample of unaffected sibling

pairs had potential value in relation to the analysis of familiality especially with regard to testing the extent to which familiality of SS choice was specific to ADHD, it was to small and contained too few SS responders. Control participants were excluded if they met the clinical thresholds for ADHD (described above) on either the CPRS–R or CTRS–R *DSM–IV* ADHD index. The relevant data were available for 131 controls—of these 19 were excluded for meeting the ADHD cut-offs used in this study ($T > 63$). The final sample therefore consisted of 112 controls.

Tasks and Measures

Clinical Evaluation

Symptom rating scales. ADHD symptoms were assessed using long versions of the CPRS–R (CPRS–R:L) and CTRS–R (CTRS–R:L). Common coexisting problems of ADHD were assessed using the Conners's Rating Scale (Anxiety and Oppositional Defiant Disorder [ODD]).

Research diagnosis. This was carried out using the PACS–Revised 2003 interview (Chen & Taylor, 2006), and the CPRS–R and CTRS–R. The PACS is a semistructured interview to collect parent-based detailed information on children's behavior. It is divided into four sections: emotional disorders, hyperkinetic disorder, disruptive behavior problems, and additional problems. In the hyperkinetic disorder section, the interviewer asks parents to describe their child's behavior in different settings, and then rate the severity and frequency of the behavior according to previously defined criteria. The settings were chosen to represent common unstructured (e.g., watching TV, reading, playing alone), semistructured (e.g., meals, outings, shopping), or structured (e.g., home tasks, homework, getting ready) daily life situations. In this study, parents were asked to focus on examples of their children's behavior during the most recent medication-free period. A standardized diagnostic algorithm based on the *DSM–IV* criteria was applied to the information from PACS and from the teacher-rated ADHD subscale from the CPRS–R and CTRS–R to derive a subtype diagnosis. The algorithm included behavioral symptoms, age of onset, situational pervasiveness, and clinical impairment, information taken from the rating scales. In addition to the ADHD diagnosis, PACS also provides a mood and an anxiety score and a diagnosis of ODD and conduct disorder (CD) based on the *DSM–IV* criteria. Previous studies have shown high interrater reliability (product-moment correlations between .76 and .96; Chen & Taylor, 2006).

Intelligence. The vocabulary, similarities, picture completion, and block design subtests from the Wechsler Intelligence Scale for Children (Wechsler, 1991) and the Wechsler Intelligence Scale for Adults (Wechsler, 1997) were administered, and scores prorated to provide a full estimate of IQ (Sattler, 1992).

Experimental Task

MIDA. The MIDA (Kuntsi et al., 2001) was used to measure the participants' preference for SS over LL alternatives. In this task participants are presented with a spaceship game-like environment in which they have control over a space battleship that has the task of shooting down enemy cruisers to defend their spaceship and win points. In each trial the child had to choose between two options, firing at a single *cruiser* that is presented first (the SS: scoring 1 point) or waiting to fire at two *cruisers* that come later (LL: 2 points). The SS reward is presented after 2 s and the LL reward after 30 s. Following identical written instructions for all sites, children were told that they would be allowed only one shot at the spaceship targets per trial (a "mission" for the child) and that they could either shoot at the first target and get 1 point or wait for the two cruisers and get 2 points. Children were also informed that there would be 20 trials, and 20 small counters were placed on a board by the side of the computer in the child's sight; the experimenter took away one counter after each trial to remind the child about the number of trials remaining.

The inclusion of two different conditions allowed us to test the three predictions relating to the contribution of IDIR and DAv to SS choice. In the no-postRD condition, the two targets were presented in the way described above and the next trial followed on immediately after the participant had secured either the SS or LL. Variation in the trial length was therefore determined by the length of the prereward delay for the chosen option. In the postRD condition, the trial length was equalized for the SS and LL by including a period of delay after the delivery of the reward (2 s for the LL or 30 s for the SS option). There was therefore always 32 s of TD (combined pre- and postdelay) per trial. After receiving the instructions, children were given three practice trials before each condition. The order of presentation of the two conditions was counterbalanced. A reward was given (after each trial) in the form of points that could be exchanged for small prizes at the end of the whole session. The dependent variable was the percentage of times that participants chose the LL, with lower values indicating a preference for choosing SS.

Procedure

Families from the clinical sample were invited to research centers for cognitive assessment and parent interview as part of the IMAGE research protocol. Families were required to withdraw any medication the probands were taking for at least 48 hr before they came to the testing session and, wherever possible, preferably for a week. Control participants were administered the MIDA task and the intelligence assessment task at one session in a separate room either at the research center or at their school environment.

Analytical Strategy

There were three analytical phases:

1. *Case-control differences*: This analysis compared 112 controls with 416 ADHD cases (360 probands plus 56 affected siblings). All children with any diagnosis of ADHD were included as preliminary analysis showed no difference by subtype for either MIDA condition, $F_{\mathrm{NoPRD}}(2, 413) = .60, p = .55$; $F_{\mathrm{PRD}}(2, 413) = .93, p = .39$. The planned analysis for this study was repeated measures analysis of covariance (ANCOVA) using a full general linear model. The between-subject independent variables being group (ADHD versus controls) and gender, the within-subject factor was condition (PRD versus no-PRD). The dependent variable was the proportion of LL choices. Covariates were to be introduced where characteristics differed between the two groups (i.e., IQ, age, ODD, and anxiety). Initial exploration of the data showed a *J*-shaped distribution with around half of children showing no sign of DAv (49.1% in the no-postcon-

dition and 60.4% in the postcondition always choosing LL). Although the use of the analysis of variance (ANOVA) model assumes normality of distribution, Monte Carlo studies with large samples have shown that it is robust to most breeches of this assumption (Glass, Peckham, & Sanders, 1972). Preliminary analyses were carried out to test that this was true in the current study. To do this we compared the results for the simple main effects and interaction term from a parametric analysis (i.e., repeated measures two-way ANOVA) with a nonparametric approach using a combination of different univariate comparisons (Wilcoxon signed-ranks test and Mann–Whitney *U* tests). In this analysis the interaction test was estimated by creating a difference score by subtracting the percentage of LL choices made in the no-postRD from the percentage made in the postRD. The same pattern and magnitude of statistical significance was found for these two analyses (see Appendix 1). It was decided therefore to proceed with the original analytical strategy given its obvious advantages in terms of modeling mixed, within- and between-subject interactions and the ability to control for confounding variables within the same model. Given the wide spread of ages within the population and the possibility that age might be a significant factor in moderating the Group and Group × Condition effects, age was introduced as a fourth independent variable into the analysis. ODD and anxiety scores were used as covariates and were taken from the CPRS–R and CTRS–R as PACS data was not available for controls.

2. *Characterizing SS and LL responder subgroups*: This analysis was carried out on all the 415 ADHD cases (probands and affected siblings irrespective of subtype). The comparison of the SS and LL responder ADHD cases employed both univariate and multivariate approaches. Univariate tests used either chi-square or *t* statistics depending on the nature of the data. Logistic regression was used to identify the independent contribution of the predictors. For this analysis clinical diagnoses of ODD, CD, anxiety, and depression (mood) were available as all probands and affected siblings had been administered the PACS interview.
3. *Familial basis of SS choice*: We divided 276 probands and their nonaffected siblings and controls ($N = 112$) with full MIDA data into SS and LL responder groups using the same criteria as above. Affected siblings were excluded to avoid a spurious increase in familiality due to the established link between ADHD and SS choice. Difference in the proportions of SS responders in the four groups were tested using chi-square. Potential confounding effects of background and clinical characteristics were controlled for using binary logistic regression.

Results

Do Child and Adolescent Patients With ADHD Choose the SS Reward More Than Controls? Is This Pattern Exacerbated Under the No-PostRD Delay Condition?

Table 1 shows the clinical and background characteristics for ADHD cases and controls. ADHD cases were slightly older, $t(526) = 2.81$, $p = .005$; and had a significantly lower IQ, $t(514) = 6.90$, $p < .001$ than controls. They were more likely to be male, $\chi^2(1, N = 528) = 9.03$, $p = .003$. They were also much more likely to display ODD, $\chi^2(1, N = 528) = 136.20$, $p < .001$; and anxiety, $\chi^2(1, N = 528) = 32.94$, $p < .001$. Anxiety, ODD, and IQ were entered into the planned ANOVA as covariates. Gender was already introduced as an independent variable. Table 1 also gives the proportion of choice for LL under the no-postRD and postRD conditions for the ADHD cases and the controls broken down by age.

Table 1
Clinical and Background Characteristics and MIDA Performance of ADHD Cases and Non-ADHD Controls Broken Down by Age

	Controls[a]		ADHD[b]	
	Young	Old	Young	Old
Gender (% males)	61.2	89.6	84.8	85.7
Age (years)	8.98	13.29	8.98	13.35
	(1.42)	(1.13)	(1.42)	(1.32)
IQ	113.91	111.78	103.25	99.09
	(12.00)	(12.27)	(15.78)	(14.82)
Conners's Anxiety (% meeting cut-off)	8.2	6.4	30.6	42.0
Conners's ODD (% meeting cut-off)	2.0	7.9	67.3	67.5
MIDA data				
No-postRD (% LL)	84.76	96.50	61.40	82.99
	(26.17)	(12.91)	(33.80)	(29.29)
PostRD (% LL)	91.81	99.51	77.64	93.16
	(18.23)	(2.23)	(24.73)	(14.15)

Note. Standard deviations are in parentheses. Anxiety and ODD represent the proportion of children meeting the T > 63 cut-off on parent and teacher Conners's scales combined. MIDA = Maudsley Index of Delay Aversion; ADHD = attention deficit/hyperactivity disorder; young = less than 12 years; old = 12 years or over; ODD = oppositional defiant disorder.=; no-postRD = no postreward delay condition; postRD = postreward delay condition.
[a] $n = 112$. [b] $n = 416$.

The analysis without covariates gave a main effect of group, $F(1, 517) = 30.06$, $p < .001$; with controls choosing LL more than ADHD cases, of condition, $F(1, 517) = 40.66$, $p < .001$; with LL being chosen more under the postRD than under the no-postRD conditions, and of age, $F(1, 517) = 27.81$, $p < .001$; with adolescents choosing the LL more than younger children. The effect of gender did not reach significance, $F(1, 517) = 2.11$, $p = .114$. There was also an interaction between group and condition, $F(1, 517) = 12.45$, $p < .001$. So that although controls chose LL more than patients with ADHD under both conditions, F_{nopRD} (1, 517) = 30.90, $p < .001$; F_{pRD} (1, 517) = 15.68, $p < .001$; the group difference was twice as large for the no-postRD than the postRD equating to .027 difference in partial η^2 (.056 vs. .029, respectively); removing the postreward delay period added a moderate but highly significant reduction in preference for LL over and above that found in the postRD. There was no three-way interaction between group, condition and age, $F(1, 517) = 0.29$, $p = .586$ suggesting that the interaction between group and condition was unaffected by age group. This was confirmed by separate analyses for the two age subgroups: under 12 years, $F_{\text{group}}(1, 295) = 24.85$, $p < .001$; $F_{\text{inter}}(1, 265) = 4.76$, $p = .030$; 12 years and over,

$F_{group}(1, 222) = 11.44$, $p < .001$; $F_{inter}(1, 222) = 9.86$, $p = .002$. No other two, three or four way interactions were significant. The main effect of condition, $F(1, 490) = 10.28$, $p = .001$; group, $F(1, 490) = 13.21$, $p < .001$; and the interaction between condition and group, $F(1, 490) = 7.86$, $p = .005$ remained significant but were somewhat reduced when IQ, ODD, and anxiety were added as covariates. IQ had a significant and large effect, $F(1, 490) = 27.21$, $p < .001$. The effect of anxiety approached significance, $F(1, 490) = 2.78$, $p = .098$. There was no effect of ODD, $F(1, 490) = 0.05$, $p = .81$. This suggests that IQ and to a much lesser extent anxiety may play some role in mediating the effects of ADHD on DAv. Figure 2 plots the estimated marginal means for LL choices under the two conditions after controlling for all factors. To aide future studies we carried out hypothetical power analyses to establish how many cases would be required to show significant case-control effects under the two conditions in the two age groups given the pattern of group means and variance seen in the current study. The following figures gave 80% power to detect differences: young postRD, $N = 48$; old postRD, $N = 103$; young no-postRD, $N = 33$; old no-postRD, $N = 77$.

Can SS and LL ADHD Responders Be Differentiated From One Another in Terms of Clinical Characteristics?

Using the 50% preference for the SS reward in the no-postRD percentage cut-off 33.6% of cases ($N = 137$) and 8.9% of controls ($N = 10$) were identified as SS responders. Although, as expected the rates of SS responders identified using this definition were higher in the young age group the 3:1 ratio between cases and controls was maintained at both ages (under 12 years to 44.4 to 14.3%; 12 years and over 16.1 to 4.8%). These effects were highly significant at both ages, $\chi^2(1, N = 528) = 26.12$, $p < .001$. Given the small number of SS responders in the older group participants were collapsed across age group for the analysis of factors that predicted group membership. Table 2 reports the clinical and background characteristics for SS and LL responders. Univariate tests suggested SS responders were different from LL responders in a number of ways. They had substantially lower IQ, were younger and more likely to be male. They were also more likely to have a PACS diagnosis of CD—but not ODD. A binary logistic regression model was used to explore the independent contributions of these factors. All variables were entered in one step. IQ ($B = -.034$, Wald = 15.53, $Exp(B) = .966$, $p < .001$), age ($B = -.45$, Wald = 56.21, $ExpB = .64$, $p < .001$), and CD ($B = -.66$, Wald = 4.16, $ExpB = 1.92$, $p = .041$) made independent significant contributions to predicting group membership. The effect of gender was no longer significant ($p = .181$).

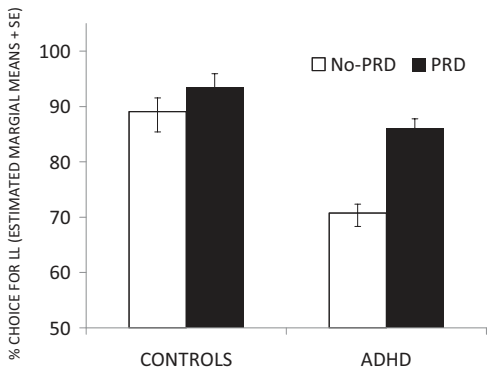

Figure 2. The percentage of choice for the large delayed reward as a function of condition and status (ADHD versus controls) adjusted for IQ, age, ODD, and anxiety.

Can SS and LL Responder Probands Be Differentiated on the Basis of Their Siblings' MIDA Performance?

Figure 3 illustrates the proportion of probands, siblings of SS responder probands, siblings of LL responders and controls, who were SS responders themselves. Siblings of SS responder probands were significantly more likely also to be SS responders, $\chi^2(1, N = 276) = 8.49$, $p = .004$. Their level of preference for SS was on a par with that seen in probands. Furthermore siblings of SS responders, $\chi^2(1, N = 186) = 12.90$, $p < .001$; but not LL responders, could be distinguished from controls, $\chi^2(1, N = 314) = 1.35$, $p = .244$. These effects persisted after controlling for ODD, anxiety, age, IQ, inattention, hyperactivity/impulsiveness, and gender in a logistic regression ($B = .75$, Wald = 4.09, $ExpB = .2.11$, $p = .043$). Once again no other factor predicted membership of the two sibling groups.

Finally we explored the possible impact of adopting other thresholds for determining subgroup membership (less than 30%, 40%, 60%, and 70% LL; Table 3). Taking a different threshold did not in general alter the effects of ADHD on SS choice or the degree of familiality as indicated by the SS choice ratio for the unaffected siblings of SS and LL responder probands. The pattern of results for comorbid internalizing and externalizing problems, scores of the inattention and the hyperactive/impulsive dimensions, age, or IQ were largely unaffected by threshold definition. The effect of gender was less marked and nonsignificant with more stringent cut-offs (30% and 40% LL).

Discussion

Children with ADHD tend to choose SS over LL in simple choice tasks more often than controls (Sonuga-Barke, 2008). The current study is the first experimental test of the relative contributions of IDIR and DAv to this choice pattern carried out with rigorously diagnosed clinical cases. The results supported a two component model with ADHD preference for SS under the postRD condition (in which the choice of SS leads only to a reduction in preRD) being accentuated under the no-postRD (in which choice of the SS reward leads to the reduction of both preRD and TD). Both IDIR and DAv appear to contribute to SS choice in ADHD on the MIDA. The very large sample and the fact that data were collected across diverse research groups working in different cultural and linguistic settings provide strong evidence for the robustness of these effects. Furthermore, analysis of the influence of covariates suggested that significant association between ADHD and DAv were not the result of differences between the ADHD cases and controls in terms of age, gender, comorbidities, or intelligence. There was also no effect of ADHD subtype (although the current study was not designed to test this).

These results therefore raise interesting questions about the origin and status of IDIR and DAv and how they come to coexist within ADHD and jointly influence SS choice. One possibility,

Table 2
Clinical and Background Characteristics of SS and LL Responders ADHD Cases

	SS responder ADHD cases[a]	LL responder ADHD cases[b]	Statistics
Age (years)	9.24 (2.58)	11.31 (2.41)	$t(414) = 8.03$, $p < .001$
IQ	98.03 (16.38)	103.44 (14.80)	$t(404) = 3.39$, $p = .001$
Sex (% males)	80.3	89.9	$\chi^2(1, N = 415) = 7.25$, $p \leq .001$
PACS Inattention	8.17 (1.13)	8.13 (1.17)	$t(414) = 0.38$, $p = .701$
PACS Hyperactive/impulsive	8.18 (1.45)	8.03 (1.31)	$t(414) = 1.06$, $p = .286$
PACS Depression (%)	7.0	10.6	$\chi^2(1, N = 415) = 1.26$, $p = .261$
PACS Anxiety (%)	46.9	39.0	$\chi^2(1, N = 415) = 2.19$, $p = .139$
PACS ODD (%)	64.1	67.2	$\chi^2(1, N = 415) = 0.546$, $p = .901$
PACS CD (%)	31.3	23.0	$\chi^2(1, N = 415) = 3.14$, $p = .076$

Note. Standard deviations are in parentheses. Clinical and IQ data were missing for one case. SS = smaller sooner; LL = larger later; ADHD = attention deficit/hyperactivity disorder; PACS = Parental Account of Children's Symptoms; ODD = oppositional defiant disorder; CD = conduct disorder.
[a] $n = 139$. [b] $n = 277$.

suggested previously, is that IDIR and DAv are elements of the same neuro-developmental mechanism and that IDIR is a developmental precursor of DAv (Sonuga-Barke, 2003; Sonuga-Barke, Brookes, et al., 2008). First, in this model IDIR is hypothesized to be a neurobiologically based trait grounded in disruptions of neuro-circuitry of the dopamine modulated, prefrontal-striatal brain reward circuits (including ventral striatum (most significant nucleus accumbens) and orbito-frontal cortex; Cardinal, Pennicott, Sugathapala, Robbins & Everitt, 2001; Cooper & Knutson, 2008; Schultz, 2002). Second, the pathway between these neurobiological alterations and IDIR is hypothesized to be mediated by deficits in the signaling of delayed rewards, coding of their incentive value and neuro-psychological processes involved in the maintenance of responding under conditions of delayed rewards (Sagvolden et al., 2005; Tripp & Wickens, 2008). Third, DAv emerges over time as delay acquires a negative affective valence for children with IDIR. This occurs as children with IDIR perform poorly in delay-rich settings and therefore come to associate such situations with failure and disappointment, especially when such poor performance leads to censure or punishment by significant others. Children with ADHD's attempt to escape and avoid such situations exacerbate impulsiveness in choice settings and inattention and overacvtivity in nonchoice setting as described in the introduction. Thus the model builds on evidence for a role of both task performance and especially task failure (Milich, 1994) and associated social/parenting factors in shaping patterns of children's motivational engagement with the environment (Gonzalez-DeHass, Willems & Holbein, 2005).

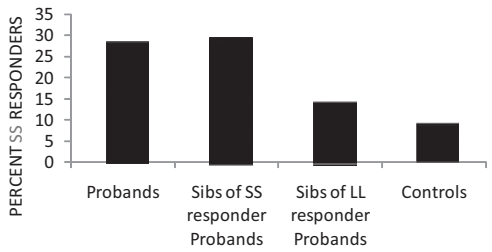

Figure 3. The proportion of siblings with and without SS responder probands that were SS responders themselves relative to the performance of probands and controls.

Longitudinal studies that allow continuities between early established IDIR and the development of DAv as well as the moderating role of the child's social environment are necessary to properly test this neuro-developmental hypothesis. However, cross sectional data of the sort discussed here may also be useful. For instance, it might be predicted on the basis of the neuro-developmental model that given its hypothesized acquired nature DAv would increase over time and adolescents should be more DAv than children. Because the current paper includes participants ranging in age from 6 to 17 years we could explore such age-related patterns. There was in fact no evidence to suggest that DAv in ADHD was greater in adolescence than childhood. On the contrary, although the effects of group as a whole and the interaction between group and condition were significant in both age groups, there was a somewhat greater differentiation between ADHD and controls in the younger group in terms of general preference for SS relative to LL in the no-postRD condition.

This result is however, difficult to interpret for a number of reasons. First, there was a large main effect of age on SS choice independent of ADHD status, with the choice of LL over SS tending to increase with age. This finding is consistent with data showing that the motivational impact of outcomes changes, as individuals mature across the life span. Small rewards become less salient in adolescence than in childhood (Wulfert et al.,

Table 3
The Results of the Comparison of SS and LL Responders With Categories Defined According To Different Cut-Offs

Definition of SS responders	<30 LL	<40 LL	<60 LL	<70 LL
Proportion of ADHD cases who were SS responders (%)	15.1	20.2	38.2	43.3
Proportion of controls who were SS responders (%)	3.1	4.6	10.8	13.8
ADHD/controls SS responder ratio	4.8	4.4	3.5	3.13
Age (years)	$t(414) = 4.23$ $p < .001$	$t(414) = 4.36$ $p < .001$	$t(414) = 6.64$ $p < .001$	$t(414) = 6.34$ $p < .001$
IQ	$t(404) = 1.86$ $p = .064$	$t(404) = 2.18$ $p = .030$	$t(404) = 3.34$ $p = .001$	$t(404) = 3.10$ $p = .002$
Sex (% males)	$\chi^2(1) = 2.30$ $p = .129$	$\chi^2(1) = 6.30$ $p = .012$	$\chi^2(1) = 7.35$ $p = .007$	$\chi^2(1) = 4.55$ $p = .033$
PACS Inattention	$t(414) = 1.31$ $p = .192$	$t(414) = 1.38$ $p = .168$	$t(414) = 0.91$ $p = .363$	$t(402) = 0.10$ $p = .896$
PACS Hyperactive/Impulsive	$t(414) = 1.00$ $p = .315$	$t(414) = 0.75$ $p = .455$	$t(414) = 0.56$ $p = .584$	$t(406) = 0.31$ $p = .756$
PACS Depression (%)	$\chi^2(1) = 2.955$ $p = .086$	$\chi^2(1) = 0.96$ $p = .328$	$\chi^2(1) = 1.70$ $p = .191$	$\chi^2(1) = 3.75$ $p = .053$
PACS Anxiety (%)	$\chi^2(1) = 0.98$ $p = .320$	$\chi^2(1) = 1.65$ $p = .199$	$\chi^2(1) = 0.99$ $p = .318$	$\chi^2(1) = 0.24$ $p = .620$
PACS ODD (%)	$\chi^2(1) = 0.638$ $p = .424$	$\chi^2(1) = 1.96$ $p = .162$	$\chi^2(1) = 0.001$ $p = .986$	$\chi^2(1) = 0.033$ $p = .855$
PACS CD (%)	$\chi^2(1) = 1.29$ $p = .256$	$\chi^2(1) = 1.10$ $p = .294$	$\chi^2(1) = 2.67$ $p = .102$	$\chi^2(1) = 1.20$ $p = .272$
Proportion of SS responder probands with unaffected SS responder siblings (%)	12.5	17.0	30.3	38.7
Proportion of non-SS responders probands with SS responder siblings (%)	6.0	8.6	18.8	21.9
Ratio of SS to non-SS responder in terms of SS responder siblings	2.08	1.97	1.61	1.76

Note. $N = 276$ for chi-square analyses. SS = smaller sooner; LL = larger later; ADHD = attention deficit/hyperactivity disorder; PACS = Parental Account of Children's Symptoms; ODD = oppositional defiant disorder; CD = conduct disorder.

2002) and the ability and/or willingness to tolerate delay to rewards grows exponentially (Bjork et al., 2004; Green et al., 1994, 1996). This could mean that DAv becomes more difficult to index as individuals grow. Longer and longer delay intervals and larger and larger rewards may be required and tasks developed for children will not be appropriate for adolescents. The highly skewed distribution of choice proportions found with adolescents suggested that this might be the case in the current study. So that although our comparison of nonparametric and parametric techniques suggested that the ANOVA models employed in the current study were robust to this breach of the assumptions for parametric statistics, a close inspection of the data from the older children suggested the operation of a ceiling effect—demonstrated by the increasingly truncated pattern of variance as the mean of LL choice for a particular data cell approached the ceiling (see Table 1). The task may have been too easy for these children with even the patients with ADHD having little difficulty choosing the LL. This leaves open the possibility that the MIDA in its current form underestimates the level of DAv in the current sample, and that, especially in older children, the real effects are greater. An examination of patterns of variance across cells in the analysis for patients less than 12 years of age suggests that ceiling effects were less of an issue in this age group. Future research with adolescents should employ tasks that either extend the delay element of LL (i.e., adjusting delay levels during the task in response to performance to maximize the differentiation between delay averse and nondelay averse; Müller, Sonuga-Barke, Brandeis, & Steinhausen, 2006) or adopt a completely different index of delay aversion (Bitsakou, Antrop, Wiersema, Sonuga-Barke, 2005). Interpreting differences between childhood and adolescents on the MIDA may be further complicated by the fact that the expression of DAv changes with age, as is the case with other symptoms of ADHD (Nutt et al., 2007). So for instance, one might expect a diminution of the behavioral manifestation of DAv accompanied perhaps by an increase in internal agitation during delay as children grow into adolescents; so that even if DAv is increasing overtime ones' ability to measure it may be more limited.

What explanations, other than IDIR/DAv, could there be for the observed effects? One possibility is that children with ADHD are especially sensitive to the economics of the sorts of choice tasks used in this study and that their pattern of responses represents an attempt to maximize some reward parameter. Although it is clear that ADHD performance under both conditions led to less reward across the whole session compared to controls another possibility is that their performance was controlled in a more local way by attempts to maximize rewards per unit of time rather than overall levels of reward. This radical and very interesting suggestion is however not consistent with the current results as it would lead to the prediction that children with ADHD should favor the LL reward under the

postRD condition, more, or at least, equally to controls—this was not the case and in fact children with ADHD sacrificed a higher reward rate to take the SS option under this condition. A second possibility is that their pattern of responses is consistent with a generalized stimulus hunger and that the preference for immediacy was driven by the desire to increase stimulation during the experiment (Zentall & Meyer, 1987). Stimulation, as a concept, is difficult to operationalize in the context of the current experiment however; in some ways it is the obverse to delay when understood in the broadest sense. Indeed, the DAv model makes predictions about the role of stimulation in those situations in which delay cannot be reduced through choices—as in the postreward delay condition. However, it differentiates temporal from nontemporal stimulation—arguing that DAv will be reduced by nontemporal (i.e., that which distracts from the passage of time) and increased by temporal (i.e., that which focuses on the passage of time) stimulation (Sonuga-Barke, 1994). Although a recent study showed that children with ADHD increased their preference for LL in the MIDA when nontemporal stimulation was added (Antrop et al., 2006), the predictions relating to the differential effects of temporal and nontemporal stimulation have not yet been tested fully and this is outside the scope of the current experiment. This therefore remains a possible explanation. Other factors such as the extent to which ADHD participants found the rewards reinforcing and the role of experimental demand should also not be ruled out. However, although these may have affected general motivation toward the task as a whole it is difficult to see how they might explain the different performance under the two conditions. Furthermore, rewards were selected by the participants themselves to maximize their putative reinforcing qualities.

The effects of ADHD on SS choice were on the whole in the moderate range. Not surprisingly the largest effect was found with the younger children under the no-postRD conditions. However, even under this condition the effects were well short of those sufficient to add diagnostic value on their own (i.e., to differentiate cases from controls reliably; Sonuga-Barke, Sergeant, et al., 2008). Despite this the effects were similar to the pooled effect sizes previously reported for the MIDA (Sonuga-Barke, Sergeant, et al., 2008) and were within the range found for other neuropsychological tasks such as those tapping executive functions (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005; Willcutt, et al., 2008). This pattern of effect sizes is consistent with the idea that ADHD is a neuro-psychologically heterogeneous disorder and that DAv/IDIR, expressed to a significant degree, affects only a (substantial) minority of patients with ADHD. In this study we estimated that about one third of the sample were SS responders overall based on an approximately 10th percentile cut-off—although this figure reached over 40% in the younger age group. These results are consistent with the proportions of SS responder participants with ADHD found in previous studies (Solanto et al., 2001). A similar pattern is seen for other neuropsychological deficits with Nigg et al. (2005) estimating that no more than 50% of ADHD children had an executive function deficit in any single executive domain, whereas only around 10% had a pervasive and severe pattern of deficit that might be predicted on the basis of recent accounts of ADHD as a executive function disorder. In fact, recent models have proposed that IDIR/DAv and executive dysfunction may mark distinctive and dissociable subgroups within the general ADHD phenotype. Initial evidence suggests that these two neuropsychological dimensions each make independent contributions to ADHD in a subgroup of children (Solanto et al., 2001; Sonuga-Barke, Dalen, & Remington, 2003; Thorell, 2007). For instance, Solanto et al. (2001) found that a preference for LL over SS on a choice task similar to that used here and performance on the Stop Signal Task (Schachar, Mota, Logan, Tannock, & Klim, 2000) were uncorrelated with each other but were moderately and significantly associated with ADHD. This sort of model of dissociated neuropsychological types has been hypothesized to be related to dysfunctions within the spatially proximate but functionally segregated circuits of the dorsal and ventral components of the thalamo-cortico-striatal loops implicated in the control of executive functions and reinforcement processes respectively (Nigg & Casey, 2005; Sonuga-Barke, 2003).

To date the characteristics of the different subgroups of SS responders have not been explored empirically. To start to address this we looked at the patterns of intelligence, age, and gender as well as comorbidity in the SS responders compared with LL responders. More interesting, the groups of SS responders had some distinctive features; they were younger (but see below), more likely to be male, more likely to have comorbid CD (although this effect was only marginally significant), and had lower IQ. The link between SS choice and IQ found in the current study was not predicted. It may suggest that these two elements may not be as independent as has previously been suggested. This result is in line with findings by Kuntsi and colleagues (2001) and Bitsakou et al., (2009). However, it is still a somewhat surprising finding. One possible explanation is that the MIDA involves decision making and that this is closely tied to IQ (Deakin, Aitken, Robbins, & Sahakian, 2004; Mazas, Finn, & Steinmetz, 2000). A second possibility is that the delay of gratification required for successful MIDA performance is influenced by socioeconomic factors and that IQ is acting as a proxy for these (Freire, Gorman, & Wessmann, 1980).

SS responders with ADHD had siblings who were more likely to be SS responders themselves. This finding is consistent with the idea that SS choice in ADHD has a familial basis and provides some initial support for the idea that family based risk factors may be mediated by those processes of IDIR/DAv that are marked by this pattern of behavior. Behavior genetic studies using twin and adoption designs data provide compelling evidence for a genetic basis to ADHD (Mick & Faraone, 2008). Molecular genetic studies have identified a number of markers showing statistically significant associations with the ADHD diagnosis. However, the size of these effects for individual genes is typically very small and even in sum they account for only a small fraction of causal variance. The published data are therefore consistent with the view that ADHD is a highly complex and heterogeneous genetic condition, with multiple genes of very small effect implicated to different degrees across affected individuals. There are a number of promising approaches to partitioning genetic heterogeneity in ADHD (Thapar, Langley, Owen, & O'Donovan, 2007). One approach, involves indentifying phenotypic characteristics that define familial subgroups of patients affected by a specific set of genes so that a more direct mapping of specific genes to disorder can be made (Crosbie, Perusse, Barr, & Schachar, 2008; Sonuga-

Barke, Sergeant, et al., 2008). The hypothesis that SS responders represent such a familial subgroup should be tested in future research.

The current study had a number of limitations. First, it would have been very useful to compare the performance on the MIDA with other tasks to further establish the independence of cognitive and motivational factors. This was outside the scope of the current analysis. Second, the lack of a sufficient number of unaffected siblings of the controls included in the current study was a significant limitation. This meant we were unable to test whether these effects were specific to ADHD or were they are also found in SS responders in the group of nonaffected controls. Finally, a comparison of different clinical subtypes would have been valuable but the numbers of participants without a combined type diagnosis was too small to allow this.

In summary the current data provide support for the two component model of the determination of SS over LL choice in ADHD in which both IDIR and DAv contribute. Furthermore, it suggests that the SS responder ADHD subset of participants can be identified and that children in this group display a specific set of characteristics. Perhaps most important their siblings are more likely than those of non-SS responders to be SS responders themselves. This provides initial evidence that DAv may mediate family based influences in ADHD.

References

Aase, H., & Sagvolden, T. (2006). Infrequent, but not frequent, reinforcers produce more variable responding and deficient sustained attention in young children with attention-deficit/hyperactivity disorder (ADHD). *Journal of Child Psychology & Psychiatry, 47,* 457–471.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Andreou, P., Neale, B., Chen, W., Christiansen, H., Gabriels, I., Heise, A., et al. (2007). Reaction time performance in ADHD: Improvement under fast-incentive condition and familial effects. *Psychological Medicine, 37,* 1703–1716.

Antrop, I., Roeyers, H., Van Oost, P., & Buysse, A. (2003). Stimulation seeking and hyperactivity in children with ADHD. *Journal of Child Psychology & Psychiatry, 41,* 225–231.

Antrop, I., Stock, P., Verte, S., Wirsema, J. R., Baeyens, D., & Roeyers, H. (2006). ADHD and delay aversion: The influence of non-temporal stimulation on choice for delayed rewards. *Journal of Child Psychology and Psychiatry, 47,* 1152–1158.

Barkley, R. A., Edwards, G., Laneri, M., Fletcher, K., & Metevia, L. (2001). Executive functioning, temporal discounting, and sense of time in adolescents with attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). *Journal of Abnormal Child Psychology, 29,* 541–556.

Bidwell, L. C., Willcutt, E. G., DeFries, J. C., & Pennington, B. F. (2007). Testing for neuropsychological endophenotypes in siblings discordant for attention-deficit/hyperactivity disorder. *Biological Psychiatry, 62,* 991–998.

Biederman, J., Petty, C. R., Fried, R., Doyle, A. E., Spencer, T., Seidman, L. J., et al. (2007). Stability of executive function deficits into young adult years: A prospective longitudinal follow-up study of grown up males with ADHD. *Acta Psychiatrica Scandinavica, 116,* 129–136.

Bitsakou, P., Antrop, I., Wiersema, J. R., & Sonuga-Barke, E. J. S. (2005). Probing the limits of delay intolerance: Preliminary young adult data from the Delay Frustration Task (DeFT). *Journal of Neuroscience Methods, 151,* 38–44.

Bitsakou, P., Psychogiou, L., Thompson, M., & Sonuga-Barke, E. J. S. (2008). Inhibitory deficits in attention-deficit/hyperactivity disorder are independent of basic processing efficiency and IQ. *Journal of Neural Transmission, 115,* 261–268.

Bitsakou, P., Psychogiou, L., Thompson, M., & Sonuga-Barke, E. J. S. (2009). Delay aversion in attention deficit/hyperactivity disorder: An empirical investigation of the broader phenotype. *Neuropsychologia, 47,* 446–456.

Bjork, J. M., Knutson, B., Fong, G. W., Caggiano, D. M., Bennett, S. M., & Hommer, D. W. (2004). Incentive-elicited brain activation in adolescents: Similarities and differences from young adults. *Journal of Neuroscience, 24,* 1793–1802.

Brookes, K., Xu, X., Chen, W., Zhou, K., Neale, B., Lowe, N., et al. (2006). Analysis of 52 candidate genes in DSM-VI combined subtype attention deficit hyperactivity disorder: Association signals in DRD4, DAT1 and 16 other genes. *Molecular Psychiatry, 11,* 934–953.

Cardinal, R. N, Pennicott, D. R., Sugathapala, C. L., Robbins, T. W., & Everitt, B. J. (2001). Impulsive choice induced in rats by lesion of the nucleus accumbens core. *Science, 292,* 2499–2501.

Castellanos, X., Sonuga-Barke, E. J. S., Tannock, R., & Milham, M. (2006). Characterising cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Science, 10,* 117–123.

Chen, W., & Taylor, E. (2006). Parental account of children′s symptoms (PACS), ADHD phenotypes and its application to molecular genetic studies. In R. D. Oades (Ed.), *Attention-deficit/hyperactivity disorder and the hyperkinetic syndrome. Current ideas and ways forward* (pp. 3–20). New York: Nova Science.

Chen, W., Zhou, K., Sham, P., Franke, B., Kuntsi, J., Campbell, D., et al. (2008). *DSM–IV* combined type ADHD shows familial association with sibling trait scores: A sampling strategy for QTL linkage. *American Journal of Medical Genetics B: Neuropsychiatric Genetics*.

Coghill, D., Nigg, J., Rothenberger, A., Sonuga-Barke, E., & Tannock, R. (2005). Whither causal models in the neuroscience of ADHD? *Developmental Science, 8,* 105–114.

Conners, C. K., Sitarenios, G., Parker, J. D., & Epstein, J. N. (1998a). The revised Conners Parent Rating Scale (CPRS–R): Factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psychology, 26,* 257–268.

Conners, C. K., Sitarenios, G., Parker, J. D., & Epstein, J. N. (1998b). Revision and restandarization of the Conners Teacher Rating Scale (CTRS–R): Factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psychology, 26,* 279–291.

Cooper, J. C., & Knutson, B. (2008) *Valence and salience contribute to nucleus accumbens activation NeuroImage, 39,* 538–547.

Crosbie, J., Perusse, D., Barr, C. L., & Schachar, R. J. (2008) *Neuroscience and Biobehavioral Reviews, 32,* 40–55.

Dalen, L., Sonuga-Barke, E. J. S., & Remington, R. E. (2004). Inhibitory deficits, delay aversion and preschool AD/HD. Implications for the dual pathway model. *Neural Plasticity, 11,* 1–11.

Deakin, J., Aitken, M., Robbins, T., & Sahakian, B. J. (2004). Risk taking during decision-making in normal volunteers changes with age. *Journal of the International Neuropsychological Society, 10,* 590–598.

Doyle, A. E., Willcutt, E., Seidman, L. J., Bierman, J., Chouinard, V.-A., Silva, J., et al. (2005). Attention-deficit/hyperactivity disorder endophenotypes. *Biological Psychiatry, 57,* 1324–1335.

Feifel, D. (1996). Attention-deficit hyperactivity disorder in adults. *Postgraduate Medicine, 100,* 207–211.

Freire, E., Gorman, B., & Wessmann, A. E. (1980). Temporal span, delay of gratification, and childrens socioeconomic status. *Journal of Genetic Psychology, 137,* 247–255.

Glass, G. V., Peckham, P. D., & Sanders, J. R. (1972). Consequences of failure to meet assumptions underlying the fixed effects analysis of variance and covariance. *Review of Educational Research, 42,* 23–288.

Gonzalez-DeHass, A., Willems, P, & Holbein, M. (2005). Examining the Relationship Between Parental Involvement and Student Motivation. *Educational Psychology Review, 17,* 99–123.

Green, L., Fry, A. F., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. *Psychological Science, 5,* 33–36.

Green, L., Myerson, J., Lichtman, D., Rosen, S., & Fry, A. F. (1996). Temporal discounting in choice between delayed rewards: The role of age and income. *Psychology and Aging, 11,* 79–84.

Hinshaw, S. P. (2003). Roots of mental illness in children. *Annals of the New York Academy of Sciences, 1008,* 149–159.

Hoerger, M. L., & Mace, F. C. (2006). A computerized test of self-control predicts classroom behaviour. *Journal of Applied Behaviour Analysis, 39,* 147–159.

Kaplan, R. F., & Stevens, M. C. (2002). A review of adult attention deficit/hyperactivity disorder: A neuropsychological and neuroimaging perspective. *CNS Spectrums, 7,* 355–362.

Kuntsi, J., Oosterlaan, J., & Stevenson, J. (2001). Psychological mechanisms in hyperactivity: I. Response inhibition deficit, working memory impairment, delay aversion, or something else? *Journal of Child Psychology and Psychiatry, 42,* 199–210.

Kuntsi, J., Rogers, H., Swinard, G., Börger, N., Van Der Meere, J., Rijsdijk, F., et al. (2006). Reaction time, inhibition, working memory and delay aversion performance: Genetic influences and their interpretation. *Psychological Medicine, 36,* 1613–1624.

Kuntsi, J., & Stevenson, J. (2001). Psychological mechanisms in hyperactivity: II. The role of genetic factors. *Journal of Child Psychology and Psychiatry, 42,* 211–219.

Lasky-Su, J., Banaschewski, T., Buitelaar, J., Franke, B., Brookes, K., Sonuga-Barke, E., et al. (2007). Partial replication of a DRD4 association in ADHD individuals using a statistically derived quantitative trait for ADHD in a family-based association test. *Biological Psychiatry, 62,* 985–990.

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.

Mazas, C. A., Finn, P. R., & Steinmetz, J. E. (2000). Decision-making biases, antisocial personality, and early-onset alcoholism. *Alcoholism, Clinical and Experimental Research, 24,* 1036–1040.

Mick, E., & Faraone, S. V. (2008). Genetics of attention deficit hyperactivity disorder. *Children and Adolescent Psychiatric Clinics of North America, 17,* 261–284.

Milich, R. (1994). The response of children with ADHD to failure—If at first you don't succeed, do you try, try again. *School Psychology Review, 23,* 11–28.

Müller, U., Sonuga-Barke, E. J. S., Brandeis, D., & Steinhausen, H.-C. (2006). Online measurement of motivational processes: Introducing the Continuous Delay Aversion Test (ConDAT). *Journal of Neuroscience Methods, 151,* 45–51.

Nigg, J. T. (2005). Neuropsychological theory and findings in ADHD: The state of the field and salient challenges for the coming decade. *Biological Psychiatry, 57,* 1424–1435.

Nigg, J. T. (2006). *What causes ADHD? Understanding what goes wrong and why.* New York: Guilford.

Nigg, J. T., & Casey, B. J. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Development & Psychopathology, 17,* 785–806.

Nigg, J. T., Willcut, E., Doyle, A. E., & Sonuga-Barke, E. J. S. (2005). Causal heterogeneity in attention-deficit/hyperactivity disorder: Do we need neuropsychologically impaired subtypes? *Biological Psychiatry, 57,* 1224–1230.

Nutt, D. J., Fone, K., Asherson, P., Bramble, D., Hill, P., Matthews, K., et al. (2007). Evidence-based guidelines for management of attention-deficit/hyperactivity disorder in adolescents in transition to adult services and in adults: Recommendations from the British Association for Psychopharmacology. *Journal of Psychopharmacology, 21,* 10–41.

Pennington, B. F. (2006). From single to multiple-deficit models of developmental disorders. *Cognition, 101,* 385–413.

Sagvolden, T., Aase, H., Zeiner, P., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. *Behavioral Brain Research, 94,* 61–71.

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. *Behavioural and Brain Sciences, 28,* 397–419.

Sattler, J. M. (1992). *Assessment of children: WISC–III and WPPSI–R supplemental.* San Diego: CA: Jerome M. Sattler.

Schachar, R., Mota, V. L., Logan, G. D., Tannock, R., & Klim, P. (2000). Confirmation of an inhibitory control deficit in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology, 28,* 227–235.

Scheres, A., Dijkstra, M., Ainslie, E., Balkan, J., Reynolds, B., Sonuga-Barke, E., et al. (2006). Temporal and probabilistic discounting of rewards in children and adolescents: Effects of age and ADHD symptoms. *Neuropsychologia, 44,* 2092–2103.

Schultz, W. (2002). Getting formal with dopamine and reward. *Neuron, 36,* 241–263.

Schweitzer, J. B., & Sulzer-Azaroff, B. (1995). Self-control in boys with attention deficit hyperactivity disorder. Effects of added stimulation and time. *Journal of Child Psychology and Psychiatry, 356,* 671–686.

Scime, M., & Norvilitis, J. M. (2006). Task performance and response to frustration in children with attention deficit hyperactivity disorder. *Psychology in the Schools, 43,* 337–386.

Sergeant, J. A., Willcutt, E., & Nigg, J. (2008). How clinically functional are executive function measures of ADHD? In D. Shaffer, E. Leibenluft, L. A. Rohde, P. Sirovatka, & D. A. Regier (Eds.), *Externalizing disorders of childhood: Refining the research agenda for* DSM–V. Arlington, VA: American Psychiatric Association.

Solanto, M. V., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G. D., Wigal, T., et al. (2001). The ecological validity of delay version and response inhibition as measures of impulsivity in ADHD: A supplement to the NIMH multimodal treatment study of ADHD. *Journal of Abnormal Child Psychology, 29,* 215–228.

Solanto, M. V., Gilbert, S. N., Raj, A., Zhu, J., Pope-Boyd, S., Stepak, B., et al. (2007). Neurocognitive functioning in AD/HD, predominantly inattentive and combined subtypes. *Journal of Abnormal Child Psychology, 35,* 729–744.

Sonuga-Barke, E. J. S. (1994). On dysfunction and function in psychological theories of childhood disorder. *Journal of Child Psychology and Psychiatry, 35,* 801–815.

Sonuga-Barke, E. J. S. (2002). Psychological heterogeneity in ADHD: A dual pathway model of motivation and cognition. *Behavioural Brain Research, 130,* 29–36.

Sonuga-Barke, E. J. S. (2003). The dual pathway model of ADHD. An elaboration of nero-developmental characteristics. *Neuroscience & Behavioral Reviews, 27,* 593–604.

Sonuga-Barke, E. J. S. (2008). What role, if any, should markers of motivational dysfunction play in the diagnosis of attention deficit hyperactivity disorder? In L. Rohde, D. Shaffer, & J. Rappaport (Eds.), *The future of psychiatric diagnosis: Externalising childhood disorder.* Washington, DC: American Psychiatric Association.

Sonuga-Barke, E. J. S., Brookes, K. J., Buitelaar, J., Anney, R., Bitsakou, P., Baeyens, D., et al. (2008). Intelligence in *DSM–IV* combined type attention-deficit/hyperactivity disorder is not predicted by either dopamine receptor/transporter genes or other previously identified risk alleles for attention-deficit/hyperactivity disorder. *American Journal of Medical Genetics B: Neuropsychiatric Genetics, 147*B, 316–319.

Sonuga-Barke, E. J. S., Dalen, L., & Remington, R. E. R. (2003). Do delay aversion and inhibitory deficits make distinct contributions to pre-school AD/HD. *Journal of the American Academy of Child & Adolescent Psychiatry, 42,* 1335–1342.

Sonuga-Barke, E. J. S., De Houwer, J., De Ruiter, K., Azensten, M., & Holland, S. (2004). ADHD and the capture of attention by briefly exposed delay-related cues. Evidence from a conditioning paradigm. *Journal of Child Psychology and Psychiatry, 45,* 274–283.

Sonuga-Barke, E. J. S., Sergeant, S., Nigg, J., & Willcutt, E. (2008). Executive dysfunction and delay aversion in ADHD: Nosological and diagnostic implications. *North American Clinics in Child & Adolescent Psychiatry, 17,* 367–384.

Sonuga-Barke, E. J. S., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion: I. The effects of delay on choice. *Journal of Child Psychology and Psychiatry, 33,* 387–398.

Thapar, A., Langley, K., Owen, M. J., & O'Donovan, M. C. (2007). Advances in genetic findings on attention deficit hyperactivity disorder. *Psychological Medicine, 37,* 1681–1692.

Thorell, L. (2007). Do delay aversion and executive function deficits make distinct contributions to the functional impact of ADHD symptoms: A study of early academic skill deficits. *Journal of Child Psychology & Psychiatry, 48,* 1061–1070.

Tripp, G., & Alsop, B. (2001). Sensitivity to reward delay in children with attention deficit hyperactivity disorder (ADHD). *Journal of Child Psychology & Psychiatry, 42,* 691–698.

Tripp, H., & Wickens, J. R. (2008). Dopamine transfer deficit: A neurobiological theory of altered reinforcement mechanisms in ADHD. *Journal of Child Psychology and Psychiatry, 49,* 691–704.

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children.* London: Psychological Corporation.

Wechsler, D. (1997). *Wechsler Intelligence Scale for Adults.* London: Psychological Corporation.

Wiersema, R., van der Meere, J., Roeyers, H., Van Coster, R., & Baeyens, D. (2006). Event rate and event-related potentials in ADHD. *Journal of Child Psychology & Psychiatry, 47,* 560–567.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of ADHD: A meta-analytic review. *Biological Psychiatry, 57,* 1336–1346.

Willcutt, E. G., Sonuga-Barke, E. J. S., Nigg, J. T., & Sergeant, J. A. (2008). Recent developments in neuropsychological models of childhood psychiatric disorders. In T. Banaschewski, & L. A. Rhode (Eds.), *Biological child psychiatry. Recent trends and developments. Advances in biological psychiatry.* Basel, Switzerland: Karger.

Wulfert, E., Block, J. A., Santa Ana, E., Rodríguez, M. L., & Colsman, M. (2002). Delay of gratification: Impulsive choices and problem behaviors in early and late adolescence. *Journal of Personality, 70,* 533–552.

Zentall, S. S., & Meyer, M. J. (1987). Self-regulation of stimulation for ADD–H children during reading and vigilance task performance. *Journal of Abnormal Child Psychology, 15,* 519–536.

Appendix

Comparison of Parametric and Nonparametric Estimates of Effects

	Effect group	Effect condition	Group × Condition
Nonparametric	Mann-Whitney U: $Z = 7.57$ $p < .001$	Wilcoxin SRT: $Z = 6.69$ $p < .001$	Mann-Whitney U: $Z = 3.59$ $p < .001$
Parametric (t test)	$t(526) = 6.78$ $p < .001$	$t(526) = 11.47$ $p < .001$	$t(526) = 3.62$ $p < .001$
Parametric ANOVA	$F(1, 526) = 46.03$ $p < .001$	$F(1, 526) = 54.81$ $p < .001$	$F(1, 472) = 13.15$ $p < .001$

Note. The table shows the equivalence of parametric and nonparametric estimates of the main effects of group (ADHD versus controls), condition (no-postRD and postRD) and interaction (Group × Condition). For the univariate tests, both parametric (t test) and nonparametric (U test) were used: The interaction term was estimated by using a difference score (preference for LL under no-postRD—preference for LL under postRD). The comparison of t and F tests supported the equivalence of the difference score and the interaction term. The comparison of the t/F and U confirm that given the large sample the parametric approaches were robust to the deviations from normality seen in the choice data. ADHD = attention deficit/hyperactivity disorder; LL = larger later; no-postRD = no postreward delay condition; postRD = postreward delay condition; ANOVA = analysis of variance.

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