Eine neue Methode zur neuropsychologischen Erfassung motivationaler Selbstregulation bei der Aufmerksamkeitsdefizit- / Hyperaktivitätsstörung

Müller, U

Eine neue Methode zur neuropsychologischen Erfassung motivationaler Selbstregulation bei der Aufmerksamkeitsdefizit- / Hyperaktivitätsstörung

Abhandlung

zur Erlangung der Doktorwürde

der Philosophischen Fakultät

der

Universität Zürich

vorgelegt von

Ulrich Müller

von Birmenstorf (AG)

Angenommen im Frühjahrssemester 2009 auf Antrag von

Herrn Prof. Dr. Dr. Hans-Christoph Steinhausen

Herrn Prof. Dr. Friedrich Wilkening

Zusammenfassung

Schwierigkeiten, ins Auge gefasste Ziele längerfristig zu verfolgen angesichts von konkurrierenden kurzfristigen Anreizen, gehören zu den Hauptmerkmalen der Aufmerksamkeitsdefizit- / Hyperaktivitätsstörung (ADHS). Beeinträchtigungen des Belohnungssystem des Gehirns und eine daraus erwachsende Intoleranz für Belohnungsaufschub (IB) ist eine der wichtigsten postulierten Ursachen für diese Schwierigkeiten.

Die vorliegende Doktorarbeit hatte zum Ziel, bekannte methodische Schwierigkeiten in der neuropsychologischen Messung von IB durch Entwicklung, Validierung und Anwendung eines neuen neuropsychologischen Computertests, des Continuous Delay Aversion Test (ConDAT), zu überwinden.

Der ConDAT konfrontiert die Testperson permanent mit der Entscheidung für oder gegen das Investieren von Zeit für einen sich stetig verringernden Zuwachs an Belohnung. Durch dieses Permanenzprinzip sowie durch sein nicht-materielles adaptives Belohnungssystem unterscheidet sich der ConDAT von bisherigen Paradigmen, welche auf prospektiven Entscheidungen zwischen zwei Belohnungstypen beruhen.

Reliabilität und Konstruktvalidität des Test wurden nachgewiesen, und erstmals wurde die IB über ein grosses Altersspektrum (6 bis 17 Jahre) valide neuropsychologisch gemessen. Es wurde gezeigt, dass die IB nicht nur abnimmt mit zunehmendem Alter, sondern auch bei kleinen Kindern stark zunimmt im Laufe einer Testsitzung; diese Tendenz weicht mit zunehmendem Alter einem stabilen Verlauf. Im Gegensatz zu gesunden Kindern und Jugendlichen war bei Kindern mit ADHS die IB konstant hoch, unabhängig sowohl vom Alter als auch vom zeitlichen Testverlauf, was als generalisierte und teilweise erlernte IB interpretiert wurde. [Einleitung und Zusammenfassung deutsch, drei Hauptkapitel englisch].

Abstract

Keeping long-term goals in mind when faced with a short-term gratification is a major problem in children with Attention-Deficit / Hyperactivity Disorder (ADHD). These difficulties have been attributed to a deficient neuronal reward system leading to a reduced delay tolerance (DT). The present thesis aimed at overcoming some methodological problems in measuring DT by developing, validating, and applying a new computerized neuropsychological task, the Continuous Delay Aversion Test (ConDAT).

Faced with a steadily diminishing return, the subject is permanently confronted with the decision between either waiting further for reward or terminating the trial because the amount of returned reward is not worth waiting for it any more. This permanence principle of the ConDAT and its adaptive, non-material reward system is different from common delay aversion (DA) tasks which are based on prospective decisions between a small immediate reward and a larger delayed reward.

In the present contribution, the ConDAT was shown to be reliable, valid, and particularly suited for developmental studies. In a large sample of children aged 6 to 17 years, DT increased with age but also decreased during the task. This time-on-task effect was most pronounced in young children and approached a constant performance across the task in adolescents. In contrast, children with ADHD had a low DT throughout the task independent of age and time-on-task. This ADHD-specific pattern is interpreted as a generalized DA reflecting individual negative experiences with delay situations besides well-known neurobiological abnormalities of the disorder. [Introduction and summary in German, main chapters in English].

Danksagung

Die vorliegende Doktorarbeit wurde unter der Aufsicht von Prof. Dr. Dr. Hans-Christop Steinhausen verfasst. Ich möchte ihm an dieser Stelle meinen aufrichtigen Dank aussprechen für seine stets unterstützende Haltung und das Vertrauen in meine Arbeit. Ohne seine fast immerwährende Präsenz auch aus grosser räumlicher Distanz und seine Bereitschaft, schnell und stets bereichernd auf meine Arbeit einzugehen und Kurswechsel flexibel zu akzeptieren, die ich während der gesamten Zeit mit einer grossen Selbstverständlichkeit in Anspruch nehmen durfte, wäre es nicht möglich gewesen, diese Arbeit zum jetzigen Zeitpunkt vorzulegen. Ebenfalls danken möchte ich Prof. Dr. Friedrich Wilkening für seine grosszügige Unterstützung dieser Arbeit.

Ein besonderer Dank gebührt Prof. Edmund Sonuga-Barke für seine grosse Wertschätzung der Originalität meiner Arbeit, seine wichtigen analytischen und strategischen Impulse und seine Ermutigung, den eingeschlagenen Weg weiter zu verfolgen.

Danke auch an Samuel Schönenberger, der engagiert und verlässlich wesentliche Grundsteine gelegt hat für diese Arbeit. Ausschliesslich danke ich den Kindern und Jugendlichen, welche mit Ihrer Bereitschaft, sich auf eine ungewohnte Aufgabe einzulassen, das Projekt unterstützt haben.

In zuletzt turbulenten Zeiten konnte ich mich stets auf die bedingungslose Unterstützung von Karin verlassen, welche mir zusätzliche Kraft gegeben hat, das längerfristige Ziel konsequent zu verfolgen.

Inhalt

1 Einleitung und Hintergrund

1.1 Übersicht und Ziel der Arbeit

Die vorliegende Arbeit will einen Beitrag leisten zum besseren Verständnis der Entstehung und der Wirkmechanismen der Aufmerksamkeitsdefizit-/Hyperaktivitässtörung (ADHS). Kinder mit ADHS reagieren stärker als normale Kinder auf unmittelbare Reize und können damit längerfristige Ziele schlechter verfolgen. In der Erfassung dieser Tendenzen gibt es bisher kein Verfahren, welches für neuropsychologische Entwicklungsstudien geeignet ist, d.h. einerseits über eine hohe Sensitivität entlang eines weiten Messbereichs verfügt und andererseits neuropsychologischen Anforderungen nach möglichst objektiver Erfassung genügt.

Ziel dieser Arbeit ist es, ein solches Verfahren zu entwickeln und seine Reliabilität zu prüfen (Studie 1), seine Eignung für neuropsychologische Entwicklungsstudien nachzuweisen (Studie 2) sowie seine Konstruktvalidiät bezüglich ADHS zu zeigen (Studie 3). Mit den Befunden aus diesen Studien soll zugleich die Theorie motivationaler Ursachen des ADHS weitere Stützung und Differenzierung erfahren.

1.2 Die Aufmerksamkeitsdefizit-/Hyperaktivitässtörung (ADHS)

Das Aufmerksamkeitsdefizit-/Hyperaktivitätssyndrom (ADHS) wird als Störung der Selbstregulation verstanden, aus welcher Konzentrationsprobleme, unkontrollierte körperliche Aktivität sowie impulsives Verhalten erwachsen (R. A. Barkley, 1997). Das ADHS gehört zu den häufigsten psychischen Störungen des Kindes- und Jugendalters, kommt oft zusammen mit anderen psychischen Störungen vor und führt häufig zu Beeinträchtigungen in Schule und Beruf, zu Problemen in Familie und Partnerschaft, aber auch zu vermehrten Unfällen oder antisozialem Verhalten (H.-C. Steinhausen, 2000).

1.2.1 Klinisches Bild

Ein weltweiter Vergleich der Auftretenshäufigkeiten des ADHS zeigt auf, dass die zum Teil beträchtlichen Häufigkeitsunterschiede eher auf unterschiedliche diagnostische Konzepte und Messmethoden zurückzuführen sind als auf soziokulturelle Faktoren (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). Die Prävalenz in der Schweiz von 5.2% liegt in einem mittleren Bereich und darf als realistisch betrachtet werden (H. C. Steinhausen, Winkler Metzke, Meier, & Kannenberg, 1999). Knaben sind ca. drei bis neunfach häufiger betroffen als Mädchen (H.-C. Steinhausen, 2000), jedoch wird auch angenommen, dass diese Schätzung zu hoch liegt, da Knaben ein unterschiedliches Symptomprofil aufweisen als Mädchen und dadurch häufiger in klinischen Studien erfasst werden (Gaub & Carlson, 1997).

Die Diagnose des ADHS wird auf Grund von klinischen Kriterien gestellt, welche in den zwei gängigen Diagnosesystemen International Classification of Diseases (ICD-10, Dilling, Mombour, & Schmidt, 1993) und Diagnostic and Statistic Manual (DSM-IV, American Psychiatric Association, 1994) festgelegt sind. Während die Sym-Symptomkriterien in beiden Systemen identisch und bis auf ein Symptom gleich unterteilt sind (s. Box 1), gibt es Unterschiede in der Anzahl Symptome, die für eine Diagnose verlangt werden und in den diagnostischen Subtypen. Während das ICD-10 System zwischen einer einfachen Aktivitäts- und Aufmerksamkeitsstörung und einer hyperkinetischen Störung des Sozialverhaltens unterscheidet, also gestörtes Sozialverhalten als zum Syndrom gehörend auffasst, unterscheidet das DSM-System die drei Subtypen, vorwiegend

Box 1

Symptome der Unaufmerksamkeit nach DSM-IV

1. beachtet häufig Einzelheiten nicht oder macht Flüchtigkeitsfehler bei den Schularbeiten, bei der Arbeit oder bei anderen Tätigkeiten,
2. hat oft Schwierigkeiten, längere Zeit die Aufmerksamkeit bei Aufgaben oder beim Spielen aufrechtzuerhalten,
3. scheint häufig nicht zuzuhören, wenn andere ihn / sie ansprechen,
4. führt häufig Anweisungen anderer nicht vollständig durch und kann Schularbeiten, andere Arbeiten oder Pflichten am Arbeitsplatz nicht zu Ende bringen,
5. hat häufig Schwierigkeiten, Aufgaben und Aktivitäten zu organisieren,
6. vermeidet häufig, hat eine Abneigung gegen oder beschäftigt sich häufig nur widerwillig mit Aufgaben, die längerdauernde geistige Anstrengungen erfordern,
7. verliert häufig Gegenstände, die er / sie für Aufgaben oder Aktivitäten benötigt,
8. läßt sich öfter durch äußere Reize leicht ablenken,
9. ist bei Alltagstätigkeiten häufig vergeßlich.

Symptome der Hyperaktivität/Impulsivität nach DSM-IV

1. zappelt häufig mit Händen und Füßen oder rutscht auf dem Stuhl herum,
2. steht in der Klasse oder in anderen Situationen, in denen Sitzenbleiben erwartet wird, häufig auf,
3. läuft häufig herum oder klettert exzessiv in Situationen, in denen das unpassend ist (bei Jugendlichen oder Erwachsenen kann dies auf ein subjektives Unruhegefühl beschränkt bleiben),
4. hat häufig Schwierigkeiten, ruhig zu spielen oder sich mit Freizeitaktivitäten ruhig zu beschäftigen,
5. ist häufig „auf Achse" oder handelt oftmals als wäre sie/ er „getrieben",
6. redet häufig übermäßig viel; Impulsivität,
7. platzt häufig mit den Antworten heraus, bevor die Frage zu Ende gestellt ist,
8. kann nur schwer warten, bis sie/ er an der Reihe ist,
9. unterbricht und stört andere häufig (platzt z. B. in Gespräche oder in Spiele anderer hinein).

unaufmerksamer Typ', ‚vorwiegend hyperaktiv/impulsiver Typ' und ‚kombinierter Typ' und sieht die Störung des Sozialverhaltens als eine unabhängige Störung an. Eine Diagnose darf allerdings nur gestellt werden, wenn zusätzliche Bedingungen erfüllt sind: Einige Symptome müssen erstmals vor dem Alter von Sieben Jahren aufgetreten sein; die Symptome dürfen nicht nur an einem Ort auftreten und müssen zu Beeinträchtigungen im Alltag führen; die Symptome dürfen weiter nicht durch andere Psychische Störungen besser erklärt werden.

Zusätzlich zu den Hauptsymptomen treten sehr häufig Begleitstörungen auf, am häufigsten eine Störung mit oppositionellem Trotzverhalten (bei mehr als der Hälfte aller Kinder mit ADHS), ferner Angststörungen, Emotionale Störungen, Lernstörungen (je bei etwa einem Viertel), oder eine Störung des Sozialverhaltens (bei etwa einem Fünftel), wobei Kombinationen verschiedener Störungen nicht selten sind (Biederman, 2005). Insgesamt ist das klinische Bild von ADHS uneinheitlich sowohl bezüglich des Schweregrades als auch der vorherrschenden Symptome.

Die Beeinträchtigungen durch ein ADHS sind vielfältig. Häufig stehen Schwierigkeiten, Fertigkeiten zu Entwickeln, die für erfolgreiche Schulleistungen notwendig sind im Vordergrund. Durch ihr Verhalten werden Kinder mit ADHS oft von anderen Kindern gemieden oder sind häufig in Streits verwickelt. Vermehrte Kritik und Zurechtweisungen durch Lehrpersonen und Eltern sowie die Ablehnung seitens anderer verhindern die Entwicklung eines normalen Selbstvertrauens (Coghill, et al., 2008; Harpin, 2005).

Kinder mit ADHS haben im Durchschnitt einen niedrigeren IQ als normale Kinder (Antshel, et al., 2008; Kuntsi, et al., 2004). Die Frage, ob hauptsächlich Aufmerksamkeitsprobleme die Entwicklung der Intelligenz – oder allenfalls die Messung Intelligenz – beeinträchtigen, oder ob allenfalls gemeinsame genetische Ursachen bestehen, wie es Kuntsi et al. (2004) vorschlagen, ist nicht geklärt.

Nach einer Studie von Biedermann et al. (2000) erfüllen gut die Hälfte der in der Kindheit Betroffenen am Ende der Adoleszenz die Diagnose nicht mehr, wobei allerdings bei etwa drei Vierteln noch eine unterschwellige Anzahl von Symptomen persistieren und etwa 90% noch unter funktionellen Beeinträchtigungen leiden. Im Erwachsenenalter sind über die Hälfte der in der Kindheit diagnostizierten Personen noch auf irgendeine Art beeinträchtigt, häufig durch Probleme am Arbeitsplatz, viele Stel-

lenwechsel, aber auch durch schwierige und oft kurze Partnerschaften oder übermässigen Genuss von Genussmitteln und Drogen (Biederman, et al., 2006; Harpin, 2005).

1.2.2 Ursachen

Entsprechend der Komplexität des klinischen Bildes der ADHS sind die Ursachen und Wirkmechanismen vielfältig und trotz zunehmender Forschungsanstrengungen noch in weiten Teilen ungeklärt. Es gibt eine familiäre Häufung der Störung. Das Risiko von Eltern und Geschwistern von betroffenen Kindern, ebenfalls die Störung zu haben, ist zwei bis acht mal grösser als bei Angehörigen von nicht betroffenen Kindern (Faraone, et al., 2005). Diese familiäre Häufung lässt allerdings die Frage immer noch offen, wieweit Umweltfaktoren und wieweit Vererbung als Ursache in Frage kommen.

Einen klaren Hinweis für genetische Ursachen ergibt sich dagegen aus der in zahlreichen Zwillings- und Adoptionsstudien gemessenen mittleren Erblichkeit von 76%. Diese besagt, dass Dreiviertel der Variabilität des ADHS in der Bevölkerung auf genetische Unterschiede zurückgeführt werden kann und beruht auf angenommen statistischen Unterschieden zwischen der genetischen Ähnlichkeit von eineiigen und zweieiigen Zwillingen sowie auf der Annahme, dass die genetischen Ausstattung und die Umwelt bei eineiigen zusammen aufgewachsenen Zwillingen identisch ist (Faraone, et al., 2005).

Molekulargenetische Studien haben gezeigt, dass die Kombination kleiner Effekt von mehrere Genen für das Zustandekommen eines ADHS verantwortlich sein muss (Brookes, et al., 2006; Faraone & Doyle, 2001). Kopplungsstudien, welche Regionen auf Chromosomen identifizieren, welche von zwei genetisch verwandten Personen mit ADHS häufiger in der gemeinsamen Variante auftreten als andere Regionen, haben zwar verschiedene Regionen identifiziert, die mit ADHS in Verbindung stehen könnten, jedoch besteht zwischen diesen Regionen und den gefundenen Kandidatengenen, wie auch zwischen den Ergebnissen verschiedener Studien wenig Übereinstimmung (Willcutt, 2008).

Obwohl das genaue Zusammenspiel zwischen einzelnen Genen und deren Wechselwirkungen untereinander und mit der Umwelt im Detail noch ungeklärt ist, weiss man, dass die wichtigste identifizierten Gene, welche das Risiko für ein ADHS beeinflussen, den Transport und der Wiederaufnahme von

Neurotransmittern im Gehirn, vorwiegend Dopamin und Serotonin regulieren (Biederman & Faraone, 2005).

Obwohl genetische Faktoren wahrscheinlich die grössere Rolle spielen bei der Entstehung der ADHS, sind auch verschiedene Umweltfaktoren identifiziert worden, welche das Risiko, eine ADHS zu entwickeln, erhöhen. Einerseits sind dies biologische und biologisch vermittelte Umweltfaktoren, wie Komplikationen während Schwangerschaft und Geburt, niedriges Geburtsgewicht infolge verfrühter Geburt, Rauchen und übermässiger Alkoholkonsum der Mutter während der Schwangerschaft (Milberger, Biederman, Faraone, Guite, & Tsuang, 1997; Willcutt, 2008). Andererseits finden sich in Familien von Kindern mit ADHS vermehrt auch psychosoziale Auffälligkeiten wie familiäre Konflikte, ein verminderter Zusammenhalt innerhalb der Familie und elterliche, vorwiegend müttlerliche, psychische Auffälligkiten (Biederman, et al., 1995). In derselben Studie wurde nachgewiesen, dass mit steigender Anzahl der in den klassischen Untersuchungen von Rutter et al. (1975a; 1975b) gefundenen familiären Risikofaktoren wie elterliche Zerstrittenheit, niedriger Sozialstatus, grosse Kinderzahl, elterliche Kriminalität, psychische Störungen der Mutter sowie Aufwachsen bei Pflegeeltern die Wahrscheinlichkeit, ein ADHS zu entwickeln, steigt.

1.2.3 Pathophysiologie

Neurotransmitterstörung

Die Wirksamkeit von dopaminerg wirkenden Stimulanzien in der Behandlung des ADHS haben ein wichtiges Argument geliefert für die Annahme einer beeinträchtigen Übertragung von Signalen zwischen Nervenzellen (Gainetdinov, et al., 1999). Ausser Dopamin sind Serotonin und Noradrenalin die wichtigsten Neurotransmitter, welche die Symptomatik von ADHS beeinflussen (Biederman & Spencer, 1999; Gainetdinov, et al., 1999; Himelstein, Newcorn, & Halperin, 2000).

Es wird heute allgemein angenommen, dass die Symptome des ADHS hauptsächlich auf eine Dysregulierung von neuronalen Regelkreisen, welche das Frontalhirn mit subkortikalen Strukturen verbinden, zurückzuführen sind. In verschiedenen Studien wurden mittels Magnetresonanztomographie bei Kindern mit ADHS Hinweise gefunden auf ein verringertes Volumen des frontalen, insbesondere des

präfrontalen Cortex, der Basalganglien, des Kleinhirns sowie des gesamten Gehirns (Krain & Castellanos, 2006). In Studien mit bildgebenden Verfahren zu funktionellen Veränderungen des Gehirns bei ADHS wurden vorwiegend Veränderungen im präfrontalen Cortex, im anterioren Cingulum, in den Basalganglien sowie im Cerebellum nachgewiesen (Arnsten, 2006; Seidman, Valera, & Makris, 2005).

Beeinträchtigte Exekutivfunktionen

Eine der bis heute wichtigsten Theorien legt nahe, dass die Symptome des ADHS auf eine primäre Dysfunktion exekutiver Funktionen zurückzuführen sind (R. A. Barkley, 1997). Exekutivfunktionen werden definiert als auf sich selbst bezogene Verhaltensweisen, welche wir einsetzen, um unser Verhalten zu steuern und damit die eigene Zukunft zu verändern, also letztlich als eine Gruppe von Prozesse der Selbstregulation (Russell A. Barkley, 2004). Bei Kindern mit ADHS wurden vielfach Beeinträchtigungen in den zu den Exekutivfunktionen gezählten Bereichen Antworthemmung, Vigilanz, Arbeitsgedächtnis und Planung festgestellt (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Gemäss Barkleys (2004) Modell kommen Defiziten in der Impulshemmung, d.h.die Fähigkeit, ein bereits aktivierte Antwort auf ein Ereignis zu hemmen, so dass eine Verzögerung der Antwort erfolgt, eine herausragende und übergeordnete Rolle zu.

Beeinträchtigtes Belohnungssystem

Neure Untersuchungen haben jedoch gezeigt, dass ein beträchtlicher Teil von Kindern und Jugendlichen mit einem ADHS über normale Exekutivfunktionen verfügen (Willcutt, 2008), und demnach auch andere Ursachen für die Störung bestehen müssen. Insbesondere wurde vielfach nachgewiesen, dass im Gegensatz zu kognitiven Ursachen, zu welchen die Exekutivfunktionen gezählt werden, auch motivationale Ursachen eine Rolle spielen, insbesondere ein Unterschied in der Wirksamkeit von positiven oder negativen Verstärkern, von Belohnungen und Bestrafungen zwischen normalen Kindern und Kindern mit ADHS (Luman, Oosterlaan, & Sergeant, 2005).

Kinder mit einem ADHS brauchen höhere Verstärkungsraten als Kontrollkinder (Haenlein and Caul (1987), Verstärker müssen bei Ihnen entweder näher beim Response liegen oder stärker sein als bei Kontrollkindern (Sagvolden & Sergeant, 1998), sie sind ungewöhnlich leicht frustrierbar (Douglas &

Parry, 1994), oder entwickeln eine erhöhte Abneigung gegen den Aufschub von Belohnung (Sonuga-Barke, Taylor, Sembi, & Smith, 1992b).

Es muss heute davon ausgegangen werden, dass Dysfunktionen in unterschiedlichen neurobiologischen Regelkreisen zur Symptomatik eines ADHS führen können, wobei meistens zwischen eher kognitive und eher motivationale Funktionen einschliessenden Regelkreisen unterschieden wird: Nigg (2001) stellt exekutiver Hemmung motivationaler Hemmung gegenüber, Castellanos et al. (2006) unterscheiden zwischen heissen und kalten Exekutivfunktionen und Sonuga-Barke (2003) von einem Pfad der Exekutivfunktionen und einem Pfad der Aversion gegen Belohungsaufschub.

1.3 Zeitbezogene Entwertung (Temporal Discounting)

Die oben angesprochenen Auffälligkeiten von Kindern mit ADHS im Bezug auf den Umfang und die zeitliche Distanz von Belohnung sind eine übersteigerte Ausprägung eines normalen Phänomens, welches nicht nur bei Menschen alltäglich vorkommt, sondern wahrscheinlich auch bei allen Tieren, welche ihr Verhalten nach positiven oder negativen Anreizen richten und über einen minimalen Zeitsinn verfügen: Je weiter ein wichtiges Ziel in der Zukunft liegt, umso mehr Anstrengung braucht die Verfolgung dieses Ziels, wenn gleichzeitig auf eine nahe liegende Belohnung verzichtet werden muss. Dieses temporal discounting genannte Phänomen (Mischel & Ayduk, 2004) wurde in den Siebzigerjahren an kleinen Kindern ausführlich mittels eines Paradigmas untersucht, welches das Kind vor die Wahl stellt, zwischen einer kleinen Belohnung (z.B. ein Plätzchen), welche sofort erfolgt und einer grösseren (z.B. zwei Plätzchen), auf welche es eine gewisse Zeit warten muss (e.g. Mischel & Baker, 1975). Anhand von Versuchen vorwiegend mit Erwachsenen zur Wahl zwischen kleinen unmittelbaren und grösseren zukünftigen hypothetischen monetären Belohnungen, welche in Grösse und Zeit variiert wurden, wurde nicht nur dieser Effekt der Entwertung mit zeitlicher Ferne beschrieben, sondern auch nachgewiesen, dass die Wirksamkeit von zukünftigen Belohnungen mit dem Alter zunimmt, jedoch mit zunehmendem Einkommen schwindet (Green, Myerson, Lichtman, Rosen, & Fry, 1996; Green, Myerson, & Ostaszewski, 1999). Walls (1973) hat gezeigt, dass bei Kindern der subjektive Wert von kleinen Spielzeugen mit dem Alter abnimmt. Ausserdem scheinen Mädchen besser als Kna-

ben in der Lage zu sein, auf eine kurzfristige Belohnung angesichts einer aufgeschobenen grösseren Belohnung verzichten zu können (Silverman, 2003).

1.4 Abneigung gegen Belohnungsaufschub (Delay Aversion)

Sonuga-Barke et al. (1992) haben in Betracht gezogen, dass für die Bevorzugung unmittelbarer kleinerer Belohnungen verschiedene Gründe verantwortlich sein könnten und durch systematisches Verändern verschiedener Parameter versucht, zwischen den drei möglichen Gründen Impulsivität, Belohnungsmaximierung und Abneigung gegen Belohnungsaufschub (Delay Aversion) zu unterscheiden (Sonuga-Barke, et al., 1992b).

Die ausschlaggebenden theoretischen Überlegungen waren wie folgt: Übereinstimmend mit verschiedenen Theorien könnte es sein, dass Kinder vermehrt die unmittelbare Belohnung wählen, weil sie auf Grund beeinträchtigter Inhibitionsprozesse (Quay, 1988) bzw. ihrer Unfähigkeit, fehlerhafte Impulse zu unterdrücken (Schachar & Logan, 1990) nicht warten können.

Zweitens ist es jedoch auch denkbar, dass dieses Verhaltensmuster eine Anpassung an situative Gegebenheiten darstellt, welche ökonomischen Regeln folgt, beispielsweise wenn durch dieses Verhalten mehr Gewinn erwirtschaftet werden kann (Belohnungsmaximierung). Ein derartiges Verhalten strebt danach, die Gesamtbelohnung zu maximieren, und zwar unabhängig von anderen Faktoren. In diesem Falle ist es auch denkbar, dass die permanente Wahl der kleinen unmittelbaren Belohnung zur grössten Gesamtbelohnung führt; nämlich dann, wenn die Testdauer nicht durch die Anzahl der Durchgänge, sondern durch die zur Verfügung stehenden Zeit bestimmt wird.

Drittens besteht die Möglichkeit, dass die Wahl der unmittelbaren Belohnung, unabhängig von anderen Faktoren, darauf abzielt, den kleinsten Gesamtaufschub, das heisst die geringste mögliche Testdauer zu wählen. Als logische Folge dieser Überlegungen entstand eine Testanordnung, welche es erlaubt, zwischen Impulsivität, Belohnungsmaximierung und Delay Aversion zu unterscheiden. Dies geschah einerseits durch Manipulation des Zeitaufschubes vor und nach der in den Durchgängen ausbezahlten Belohnung, andererseits durch Festlegung der Testdauer entweder durch die Anzahl der Durchgänge oder durch die insgesamt zur Verfügung stehende Zeit. Die getesteten Kinder mit AHDS

verhielten sich über beide Experimente hinweg weder impulsiv noch gewinnmaximierend, sondern ‚delay averse'.

1.5 Mangelnde Stimulierung

Hyperaktives Verhalten von Kindern mit ADHS ist abhängig von der Umgebung. In neuen oder besonders interessanten Situationen reduzieren sie oft ihr hyperaktives Verhalten. Umgekehrt konnte nachgewiesen werden, dass die hyperaktive Symptomatik bei ADHS über die Zeit (Alberts & Van der Meere, 1992) sowie in Umgebungen mit niedrigem Stimulationsniveau ansteigt (S. Zentall, 1975). Die Erhöhung der Hyperaktivität in einer reizarmen Umgebung hat gemäss Zentall (1975) den Zweck, einen erhöhten Stimulationsbedarf zu decken. Sonuga-Barke (1994) hat diese Theorie erweitert und zwischen temporaler und nicht-temporaler Stimulation unterschieden. Dabei wird hyperaktives Verhalten aufgefasst als nicht-temporale Stimulation, welche die subjektiv erlebte Wartezeit (delay) verkürzt.
Verschiedene neuere Untersuchungen legen nahe, dass ein stärkeres Mass an äusserer Stimulation zu einer Reduktion der ADHS-Symptomatik führt (Antrop, et al., 2006; S. S. Zentall & Zentall, 1976) bzw. dass Kinder mit ADHD in ruhigen Umgebungen ihre Aktivität erhöhen um eine optimale Stimulierung zu gelangen (Antrop, Roeyer, Oost, & Buysse, 2000).

1.6 Das Dual Pathway Modell

Das von Sonuga-Barke (2002, 2005) entwickelte und später revidierte Dual Pathway Model beschreibt ADHS als Folge zweier unterscheidbarer neuropsychologischer Prozesse bzw. Entwicklungsprozesse. Ein Pfad des Modells führt die Symptome des ADHS auf Störungen des neurokognitiven Kontrollsystems zurück, in dem exekutive Prozesse (s. oben), die auf verminderter Antworthemmung beruhen, zentral sind. Diese Prozesse werden vorwiegend durch Dopamin reguliert und laufen in Regelkreisen ab, welche dorsolateralen prefrontalen Cortex, das dorsale Neostriatum, den nuceus caudatus und den dorsomedialen Thalamus einbeziehen (Sonuga-Barke, 2005).
Der zweite Pfad führt die Symptome des ADHS auf suboptimale Belohnungsprozesse zurück, bei welchen die Assoziation zwischen gegenwärtigem Verhalten und zukünftigen Belohnungen zu

schwach ist. Dies führt zu einer verminderten Fähigkeit von in der Zukunft liegenden Anreizen, das gegenwärtige Verhalten zu beeinflussen. Die vorwiegend dopaminergen Regelkreise dieses Systems verlaufen ähnlich wie diejenigen des exekutiven Pfades, schliessen aber unterschiedliche Substrukturen (orbitofrontaler Cortex, anteriores Cingulum, ventrales Striatum, ventrales Pallidum, ventraler Thalamus) mit ein (Sonuga-Barke, 2005).

Box 2

Motivationaler Pfad des erweiterten *Dual Pathway*-Modells (Sonuga-Barke, 2005)

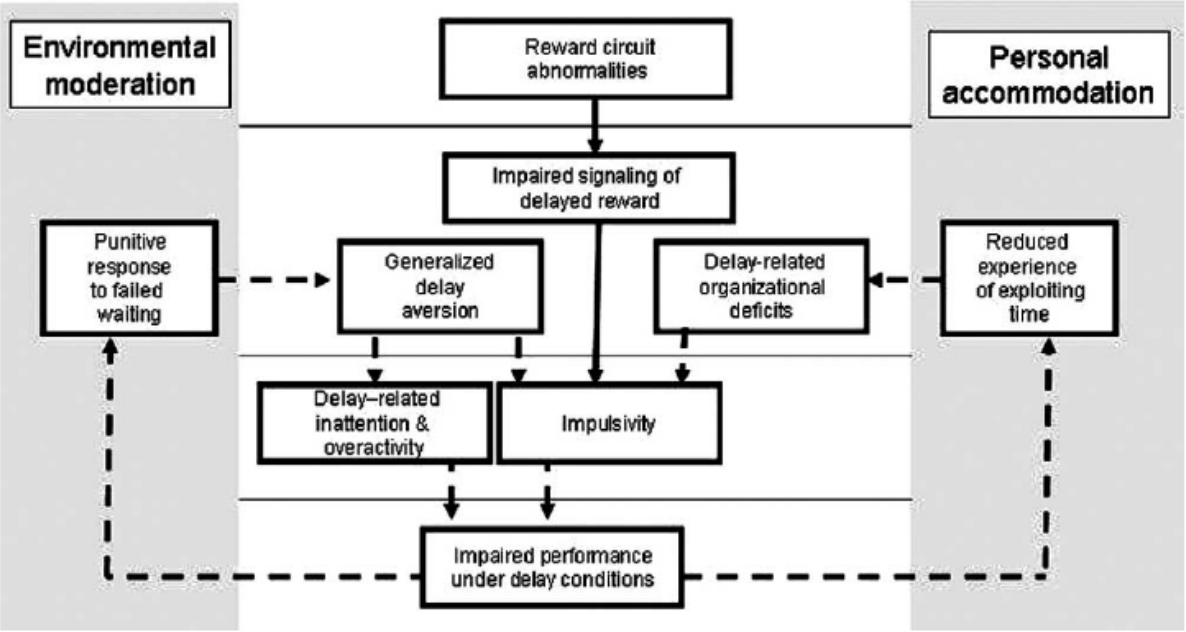

Abbildung aus (Sonuga-Barke, 2005)

Dysfunktionen in Regelkreisen des Belohnungssystems (*Reward cricuit*) führen zu einer verminderten neuronalen Repräsentation von zeitlich entfernten Belohnungsreizen (*Impaired signaling of delayed reward*). Als Folge davon werden vermehrt unmittelbare Anreize befolgt, was als Form der Impulsivität bezeichnet wird (*Impulsivity*) und zu ungenügenden Leistungen führt in Situationen, in welchen der Verzicht auf unmittelbare Belohnungen zu Gunsten in der Zukunft liegender Ziele wichtig ist.

Falls ein solches Verhalten durch die Umgebung negativ beurteilt oder bestraft wird, kann eine allgemeine, chronische Abneigung gegen Situationen entstehen, welche Belohnungsaufschub erfordern (*Generalized delay aversion*). Als Folge davon werden in künftigen solchen Situationen, falls sie nicht vorzeitig verlassen werden können, selbststimulierende Strategien ergriffen, um die subjektiv erlebte Zeit zu verkürzen: entweder wird die Aufmerksamkeit vermehrt vorhandenen Reizen in der Umgebung zugewandt (*Delay related Inattention*) oder es werden durch motorische Aktivität Wahrnehmungen erzeugt, welche die wahrgenommene Zeit ‚füllen' (*Delay realted Hyperactivity*).

Wenn Kinder im Umgang mit Belohnungsaufschub mehrheitlich scheitern, d.h. keine Fertigkeiten entwickeln können, angesichts ihrer Einschränkungen solche Situationen zu meistern, kann sich die Tendenz zu einem Verhalten, welches vorwiegend durch unmittelbare Anreize gesteuert wird (*Impulsivity*) weiter verstärken.

Das erweiterte Modell (siehe Box 2) integriert zusätzlich Entwicklungs- und Umweltfaktoren. Auf dem motivationalen Pfad können ablehnende oder bestrafende Reaktionen der Bezugspersonen auf das Verhalten in Situationen, in denen Toleranz für aufgeschobene Belohnungen erwartet wird, zu einer generalisierten Abneigung gegen Belohnungsaufschub führen, welche sekundär die ADHS-Symptomatik verstärkt. Zusätzlich kann sich die Symptomatik verschlimmern, wenn Fertigkeiten im Umgang mit solchen Situationen nicht entwickelt werden können.

1.7 Erfassung von Delay Aversion

Die frühen Experimente von Mischel (1974; 1975; Mischel, Grusec, & Masters, 1969) habe die Neigung zu unmittelbaren kleinen Belohnungen im Vergleich zu aufgeschobenen grösseren Belohnungen gemessen durch Zählen, in wie vielen Entscheidungssituationen sich ein Kind für die eine oder andere Variante entschieden hat. Das in diesen Tests eingesetzte Paradigma wurde in seinen Grundzügen in vielen Studien zur Erforschung der Abneigung gegen Belohnungsaufschub bei Kindern mit ADHS verwendet (e.g. Antrop, et al., 2006; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986; Scheres, et al., 2006; Schweitzer & Sulzer-Azaroff, 1995; Sonuga-Barke, Taylor, & Heptinstall, 1992a; Tripp & Alsop, 2001), und in den meisten Fällen wurde eine vermehrte Tendenz zur Wahl der unmittelbaren Belohnung bei Kindern mit ADHD nachgewiesen.

Durch die Einschränkung der Stichproben auf einen relativ engen Altersbereich in den meisten Studien wurde das Problem umgangen, wichtige und in ihrer Dimension grosse Einflussfaktoren zu integrieren: die stark wachsende Toleranz für aufgeschobene Belohnung mit wachsendem Alter (e.g. Green, et al., 1999) und die Abnahme des subjektiven Wertes von materiellen, insbesondere monetären Anreizen mit dem Alter (e.g. Walls, 1973). Green (1994) hat versucht, dieses Problem zu umgehen, indem er die fiktiven hohen Geldbeträge den Altersgruppen angepasst hat; allerdings geschah diese Anpassung nach subjektiver Einschätzung. Zudem haben diese Versuchsanordnungen den Nachteil, dass nicht nur die Belohnung fiktiv ist und daher von verschiedenen Personen unterschiedlich realitätsnah bewertet wurde, sondern auch der zeitliche Aufschub dieser Belohnung bloss vorgestellt ist.

In einer kürzlich veröffentlichen grossen Multicenterstudie (Marco, et al., 2008) wurden 821 Kinder mit einem neuropsychologischen Test zur Messung von Delay Aversion, dem Maudsley Index of Delay Aversion (MIDA, Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001) untersucht, welcher insofern neuropsychologischen Anforderungen genügt, als er eine weitgehend objektive quantitative Erfassung von Fähigkeiten oder Beeinträchtigungen ermöglicht (Spreen & Strauss, 1998), indem er sowohl reale Belohnungen (kleine Spielwaren oder Schreibgeräte) verwendet als den Aufschub der Belohnungsauszahlung nicht als vorgestellte Grösse einsetzt, sonder in den Testablauf integriert. Im Gegensatz zu den oben erwähnten Tests für Delay Aversion wurde in der Studie von Marco et al. (2008) Kinder mit einer grossen Altersspanne (6 bis 17 Jahre) untersucht. Die Ergebnisse haben aufgezeigt, dass der Test zwar bei jungen Kindern zwischen solchen mit ADHS und Kontrollkindern differenzieren kann, jedoch ab ca. 10 Jahren massive Deckeneffekte auftreten: in der gesamten Stichprobe habe etwa 45% der Kinder mit ADHS und 75% der Kontrollkinder den Maximalwert erreicht, also sich durchwegs für die spätere, grössere Belohnung entschieden.

Die Anforderungen an einen neuropsychologischen Test zur Messung von Delay Aversion können somit wie folgt zusammengefasst werden:

- Verzicht auf personenabhängige Einschätzungen
- Verzicht auf bloss vorgestellt Belohnungen und Zeitpunkte der Ausschüttung durch Integration beider Aspekte in den Testablauf
- Berücksichtigung der starken Entwicklung der Toleranz für Belohnungsaufschub mit dem Alter durch grossen Messbereich
- Möglichkeit zur Differenzierung zwischen verschiedenen Krankheitsgruppen durch hohe Auflösung
- Minimierung der Abnahme des Wertes der Belohnung mit dem Alter
- Quantitative, möglichst kontinuierliche Ausgabegrössen
- Erfüllung von allgemeinen Testgütekriterien wie Realität und Validität
- Allenfalls Einsetzbarkeit in neurophysiologischen Studien oder bildgebenden Verfahren

1.8 Übersicht über die drei Studien

In den folgenden Kapiteln soll dargestellt werden, welche Lösung hinsichtlich der oben formulierten Anforderungen gefunden wurde und wie das entwickelte Verfahren überprüft und angewandt wurde. Diese Kapitel, wie auch der Appendix, sind, da es sich entweder um bereits publizierte Texte (Kapitel 2) oder um zur Veröffentlichung eingereichte Texte (Kapitel 3 und 4 sowie Appendix) handelt, in Englisch, der Originalsprache der Publikationen, verfasst.

Kapitel 2 enthält eine bereits publizierte Studie (Müller, Sonuga-Barke, Brandeis, & Steinhausen, 2006) zum Hintergrund und der Methodik des Entwickelten Continuous Delay Aversion Test (ConDAT) sowie erste Pilotdaten und Angaben über die Test-Retest-Reliabilität.

In Kapitel 3 wird die methodische Eignung des Tests für Entwicklungsstudien überprüft an einer Stichprobe von 183 Schulkindern im Alter von sechs bis siebzehn Jahren. Die kritische Frage in dieser Studie betrifft die Sensitivität des Tests über den gesamten Altersbereich und damit auch das Vermeiden von Boden und Deckeneffekten. Darüber hinaus präsentiert diese Studie zum ersten Mal valide neuropsychologische Befunde über die Entwicklung von Delay Aversion von der frühen Kindheit bis zur Adoleszenz. Diese Studie wurde zur Veröffentlichung eingereicht (Müller, Sonuga-Barke, & Steinhausen, 2009).

Kapitel 4 behandelt die Frage nach der Konstruktvalidität des ConDAT (Müller, Schönenberger, Sonuga-Barke, & Steinhausen, 2009). Wie gut kann der Test indirekt eine ADHS-Diagnose verhersagen, indem er ein für die Krankheit wichtiges und bereits etabliertes Merkmal (Delay Aversion) misst und aus den Resultaten die Zugehörigkeit eines Kindes entweder zur ADHS-Gruppe oder zur Kontrollgruppe vorhersagt? Eine gute Vorhersagekraft würde nicht nur die Güte des Tests zeigen, sondern auch die Bedeutung von Delay Aversion für die Krankheit ADHS erhärten.

Im abschliessenden Kapitel 5 werden die gefundenen Ergebnisse zusammengefasst und vor dem Hintergrund der in den Studien gesetzten Ziele und aufgestellten Hypothesen sowie im Bezug auf andere Befunde auf diesem Gebiet diskutiert und hinsichtlich ihrer Konsequenzen für weitere Forschung diskutiert.

1.9 Literatur

Alberts, E., & Van der Meere, J. J. (1992). Observations of hyperactive behaviour during vigilance. *Journal of Child Psychology and Psychiatry, 33*(8), 1355-1364.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition*. Washington DC: American Psychiatric Association.

Antrop, I., Roeyer, H., Oost, P. V., & Buysse, A. (2000). Stimulation Seeking and Hyperactivity in Children with ADHD. *Journal of Child Psychology and Psychiatry, 41*(2), 225-231.

Antrop, I., Stock, P., Verte, S., Wiersema, 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 & Psychiatry & Allied Disciplines, 47*(11), 1152-1158.

Antshel, K. M., Faraone, S. V., Maglione, K., Doyle, A., Fried, R., Seidman, L., et al. (2008). Temporal stability of ADHD in the high-IQ population: results from the MGH Longitudinal Family Studies of ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry, 47*(7), 817-825.

Arnsten, A. F. (2006). Fundamentals of attention-deficit/hyperactivity disorder: circuits and pathways. *Journal of Clinical Psychiatry, 8*, 7-12.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.

Barkley, R. A. (2004). Attention-deficit/hyperactivity disorder and self-regulation: Taking an evolutionary perspective on executive functioning. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications.* (pp. 301-323). New York, NY: Guilford Press.

Biederman, J. (2005). Attention-deficit/hyperactivity disorder: a selective overview. *Biological Psychiatry, 57*(11), 1215-1220.

Biederman, J., & Faraone, S. V. (2005). Attention-deficit hyperactivity disorder. *Lancet, 366*(9481), 237-248.

Biederman, J., Faraone, S. V., Spencer, T. J., Mick, E., Monuteaux, M. C., & Aleardi, M. (2006). Functional impairments in adults with self-reports of diagnosed ADHD: A controlled study of 1001 adults in the community. *Journal of Clinical Psychiatry, 67*(4), 524-540.

Biederman, J., Mick, E., & Faraone, S. V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. *American Journal of Psychiatry, 157*(5), 816-818.

Biederman, J., Milberger, S., Faraone, S. V., Kiely, K., Guite, J., Mick, E., et al. (1995). Impact of adversity on functioning and comorbidity in children with attention-deficit hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 34*(11), 1495-1503.

Biederman, J., & Spencer, T. (1999). Attention-deficit/hyperactivity disorder (ADHD) as a noradrenergic disorder. *Biological Psychiatry, 46*(9), 1234-1242.

Brookes, K., Xu, X., Chen, W., Zhou, K., Neale, B., Lowe, N., et al. (2006). The analysis of 51 genes in DSM-IV combined type attention deficit hyperactivity disorder: association signals in DRD4, DAT1 and 16 other genes. *Molecular Psychiatry, 11*(10), 934-953.

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing cognition in ADHD: beyond executive dysfunction. *Trends in Cognitive Sciences, 10*(3), 117-123.

Coghill, D., Soutullo, C., d'Aubuisson, C., Preuss, U., Lindback, T., Silverberg, M., et al. (2008). Impact of attention-deficit/hyperactivity disorder on the patient and family: results from a European survey. *Child & Adolescent Psychiatry & Mental Health [Electronic Resource], 2*(1), 31.

Dilling, H., Mombour, W., & Schmidt, W. (Eds.). (1993). *Internationale Klassifikation Psychischer Störungen, ICD-10 Kapitel V (F)* (2 ed.). Bern: Huber.

Douglas, V. I., & Parry, P. A. (1994). Effects of reward and nonreward on frustration and attention in attention deficit disorder. *Journal of Abnormal Child Psychology, 22*(3), 281-302.

Faraone, S. V., & Doyle, A. E. (2001). The nature and heritability of attention-deficit/hyperactivity disorder. *Child & Adolescent Psychiatric Clinics of North America, 10*(2), 299-316.

Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A., et al. (2005). Molecular genetics of attention-deficit/hyperactivity disorder. *Biological Psychiatry, 57*(11), 1313-1323.

Gainetdinov, R. R., Wetsel, W. C., Jones, S. R., Levin, E. D., Jaber, M., & Caron, M. G. (1999). Role of serotonin in the paradoxical calming effect of psychostimulants on hyperactivity.[see comment]. *Science, 283*(5400), 397-401.

Gaub, M., & Carlson, C. L. (1997). Gender differences in ADHD: a meta-analysis and critical review.[erratum appears in J Am Acad Child Adolesc Psychiatry 1997 Dec;36(12):1783]. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(8), 1036-1045.

Green, L., Fry, A. F., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. *Psychological Science, 5*(1), 33-36.

Green, L., Myerson, J., Lichtman, D., Rosen, S., & Fry, A. (1996). Temporal discounting in choice between delayed rewards: The role of age and income. *Psychology and Aging, 11*(1), 79-84.

Green, L., Myerson, J., & Ostaszewski, P. (1999). Discounting of delayed rewards across the life span: Age differences in individual discounting functions. *Behavioural Processes, 46*(1), 89-96.

Haenlein, M., & Caul, W. F. (1987). Attention deficit disorder with hyperactivity: a specific hypothesis of reward dysfunction. *Journal of the American Academy of Child & Adolescent Psychiatry, 26*(3), 356-362.

Harpin, V. A. (2005). The effect of ADHD on the life of an individual, their family, and community from preschool to adult life. *Archives of Disease in Childhood, 90*(Suppl 1), i2-i7.

Himelstein, J., Newcorn, J. H., & Halperin, J. M. (2000). The neurobiology of attention-deficit hyperactivity disorder. *Frontiers in Bioscience, 5*(78), 1.

Krain, A. L., & Castellanos, F. X. (2006). Brain development and ADHD. *Clinical Psychology Review, 26*(4), 433-444.

Kuntsi, J., Eley, T. C., Taylor, A., Hughes, C., Asherson, P., Caspi, A., et al. (2004). Co-occurrence of ADHD and low IQ has genetic origins. *American Journal of Medical Genetics, Part B, Neuropsychiatric Genetics: the Official Publication of the International Society of Psychiatric Genetics. 124*(1), 41-47.

Kuntsi, J., Stevenson, J., Oosterlaan, J., & Sonuga-Barke, E. J. (2001). Test-retest reliability of a new delay aversion task and executive function measures. *British Journal of Developmental Psychology, 19*(3), 339-348.

Luman, M., Oosterlaan, J., & Sergeant, J. A. (2005). The impact of reinforcement contingencies on AD/HD: a review and theoretical appraisal.[erratum appears in Clin Psychol Rev. 2005 Jun;25(4):533]. *Clinical Psychology Review, 25*(2), 183-213.

Marco, R., Miranda, A., Schlotz, W., Melia, A., Mulligan, A., Mueller, U., et al. (2008). Delay and Reward Choice in ADHD: An Experimental Test of the Role of Delay Aversion. *Neuropsychology,* Manuscript in-press.

Milberger, S., Biederman, J., Faraone, S. V., Guite, J., & Tsuang, M. T. (1997). Pregnancy, delivery and infancy complications and attention deficit hyperactivity disorder: issues of gene-environment interaction. *Biological Psychiatry, 41*(1), 65-75.

Mischel, W. (1974). Processes in delay of gratification. In L. Berkowitz (Ed.), *Advances in experimental child psychology* (pp. 239-292). New York: Academic Press.

Mischel, W., & Ayduk, O. (2004). Willpower in a cognitive-affective processing system: The dynamics of delay of gratification. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 99-129). New York, NY: Guilford Press.

Mischel, W., & Baker, N. (1975). Cognitive appraisals and transformations in delay behavior. *Journal of Personality and Social Psychology, 31*(2), 254-261.

Mischel, W., Grusec, J., & Masters, J. C. (1969). Effects of expected delay time on the subjective value of rewards and punishments. *Journal of Personality & Social Psychology, 11*(4), 363-373.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin, 127*(5), 571-598.

Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: a systematic review and metaregression analysis.[see comment]. *American Journal of Psychiatry, 164*(6), 942-948.

Quay, H. C. (1988). Attention Deficit Disorder and the Behavioral Inhibition System: The Relevance of the Neuropsychological Theory of Jeffrey A. Gray. In L. M. Bloomingdale & J. A. Sergeant (Eds.), *Attention Deficit Disorder: Criteria, Cognition, Intervention* (pp. 117-125). Oxford: Pergamon Press.

Rapport, M. D., Tucker, S. B., DuPaul, G. J., Merlo, M., & Stoner, G. (1986). Hyperactivity and frustration: the influence of control over and size of rewards in delaying gratification. *Journal of Abnormal Child Psychology, 14*(2), 191-204.

Rutter, M., Cox, A., Tupling, C., Berger, M., Yule, W., Rutter, M., et al. (1975a). Attainment and adjustment in two geographical areas. I--The prevalence of psychiatric disorder

Attainment and adjustment in two geographical areas: I. The prevalence of psychiatric disorder. *British Journal of Psychiatry, 126*, 493-509.

Rutter, M., Yule, B., Quinton, D., Rowlands, O., Yule, W., & Berger, M. (1975b). Attainment and adjustment in two geographical areas: III--Some factors accounting for area differences. *British Journal of Psychiatry, 126*, 520-533.

Sagvolden, T., & Sergeant, J. A. (1998). Attention deficit/hyperactivity disorder--from brain dysfunctions to behaviour. *Behavioural Brain Research, 94*(1), 1-10.

Schachar, R., & Logan, G. (1990). Are hyperactive children deficient in attentional capacity? *Journal of Abnormal Child Psychology, 18*(5), 493-513.

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*(11), 2092-2103.

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 & Psychiatry & Allied Disciplines, 36*(4), 671-686.

Seidman, L. J., Valera, E. M., & Makris, N. (2005). Structural brain imaging of attention-deficit/hyperactivity disorder. *Biological Psychiatry, 57*(11), 1263-1272.

Silverman, I. W. (2003). Gender Differences in Delay of Gratification: A Meta-Analysis. *Sex Roles, 49*(9-10), 451-463.

Sonuga-Barke, E. J. (1994). On dysfunction and function in psychological theories of childhood disorder. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 35*(5), 801-815.

Sonuga-Barke, E. J. (2002). Psychological heterogeneity in AD/HD--a dual pathway model of behaviour and cognition. *Behavioural Brain Research, 130*(1-2), 29-36.

Sonuga-Barke, E. J. (2003). The dual pathway model of AD/HD: an elaboration of neuro-developmental characteristics. *Neuroscience & Biobehavioral Reviews, 27*(7), 593-604.

Sonuga-Barke, E. J. (2005). Causal models of attention-deficit/hyperactivity disorder: from common simple deficits to multiple developmental pathways. *Biological Psychiatry, 57*(11), 1231-1238.

Sonuga-Barke, E. J., Taylor, E., & Heptinstall, E. (1992a). Hyperactivity and delay aversion--II. The effect of self versus externally imposed stimulus presentation periods on memory. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 33*(2), 399-409.

Sonuga-Barke, E. J., Taylor, E., Sembi, S., & Smith, J. (1992b). Hyperactivity and delay aversion--I. The effect of delay on choice. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 33*(2), 387-398.

Spreen, O., & Strauss, E. (1998). *A Compendium of Neuropsychological Tests* (2 ed.). New York: Oxford University Press.

Steinhausen, H.-C. (2000). Klinik und Konzepte der hyperkinetischen Störungen. In H.-C. Steinhausen (Ed.), *Hyperkinetische Störungen bei Kindern, Jugendlichen und Erwachsenen* (2 ed., pp. 9-37). Stuttgart: Kohlhammer.

Tripp, G., & Alsop, B. (2001). Sensitivity to reward delay in children with attention deficit hyperactivity disorder (ADHD). *Journal of Child Psychology and Psychiatry, 42*(5), 691-698.

Walls, R. T. (1973). Delay of reinforcement development. *Child Development, 44*(3), 689-692.

Willcutt, E. G. (2008). The Etiology of ADHD: Behavioral and Molecular Approaches. In D. M. Barch (Ed.), *Handbook of Cognitive and Affective Neuroscience of Psychopathology*. London: Oxford University Press.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review. *Biological Psychiatry, 57*(11), 1336-1346.

Zentall, S. (1975). Optimal stimulation as theoretical basis of hyperactivity. *American Journal of Orthopsychiatry, 45*(4), 549-563.

Zentall, S. S., & Zentall, T. R. (1976). Activity and task performance of hyperactive children as a function of environmental stimulation. *Journal of Consulting and Clinical Psychology, 44*(5), 693-697.

2 Studie 1: Online measurement of motivational processes: Introducing the Continuous Delay Aversion Test (ConDAT)[1]

2.1 Abstract

The Continuous Delay Aversion Test (ConDAT), a new computer task for online monitoring and continuously measuring delay aversion (DA), is introduced. DA is a motivational style related to a shortened delay gradient which is proposed as a major endophenotype of attention deficit hyperactivity disorder (ADHD). It is characterised by avoiding or escaping from delay-rich situations despite the prospects of a reward. In each ConDAT trial the rapidly diminishing reward/delay ratio, which tends asymptotically towards zero, is visually presented on the computer screen. The test subject is permanently confronted with the question whether to quit or to continue the trial in the face of the deteriorating reward/time ratio. An elaborated control of stimuli and responses, including the sending of trigger codes to external recording devices, makes the task useful for neurophysiological or brain imaging experiments. Compared to existing tasks, the ConDAT is more flexible and sensitive due to its asymptotic open-ended trials and the interval-scaled output measure. Pilot data give evidence for satisfactory reliability and external validity of the task.

[1] Ueli C. Müller, Edmund J.S. Sonuga-Barke, Daniel Brandeis, Hans-Christoph Steinhausen. Journal of Neuroscience Methods 151 (2006) 45–51

2.2 Introduction

Attention-deficit/hyperactivity disorder (ADHD) is probably the most common disabling psychiatric condition in childhood (Barkley, 1998; Sergeant, 2000; Taylor, 1998). The clinical picture of the disorder itself is heterogeneous and additionally is often comorbid with other disorders (Castellanos, 1997). The main symptoms are inattentiveness, hyperactivity and impulsivity. They occur alone and/or in combination and often are accompanied by learning disabilities, oppositional defiant disorder, conduct disorder or mood disorders (for an overview see Brown et al., 2001; Paule et al., 2000; Taylor, 1998; Wilens et al., 2002). Besides a strong genetic predisposition, environmental risk factors also contribute to the risk of developing ADHD (Biederman et al., 1995; Bradley and Golden, 2001; Milberger et al., 1997).

As a genetic perspective becomes more important in ADHD research, a two-fold complexity is revealed: (1) multiple genes and their interaction contribute to the ADHD risk (Faraone et al., 2005; Kent, 2004; Shastry, 2004) and (2) the behavioural traits, which are based on diagnostic criteria, are dependent on subjective judgement, on local and historical conditions, and often are overlaid by comorbid conditions. Because of this, endophenotypes are recognised to have the potential to improve the understanding of complex psychiatric disorders, including ADHD, and provide more power for genetic analyses, as they are supposed to be more closely linked to the genetic underpinnings than symptom based definitions are (Almasy and Blangero, 2001; Castellanos and Tannock, 2002; Gottesman and Gould, 2003; Swanson et al., 1998). Endophenotypes can be conceptualized at a number of levels of analysis within the structures and processes mediating disease and distal genotype, e.g. neurophysiology, biochemistry, endocrinology, neuroanatomy, cognitive psychology, neuropsychology (Gottesman and Gould, 2003). Castellanos and Tannock (2002) postulate four main candidate ADHD-endophenotypes: deficient response inhibition (Barkley, 1997; Nigg, 2001; Oosterlaan and Sergeant, 1998), a shortened reward delay gradient (Castellanos and Tannock, 2002; Solanto et al., 2001; Sonuga-Barke, 2002; Sonuga-Barke et al., 1992a), altered temporal processing (Barkley et al., 1997; Smith et al., 2002) and working memory deficits (Barkley, 1997; Barnett et al., 2001). Following Cas-

tellanos and Tannock (2002), endophenotypes should be continuously quantifiable, predict disorder probabilistically, be closer to causative agents and be based on neuroscience.

One of these four endophenotypes, the shortened delay gradient, is the endophenotypical background for the task presented in this paper. It is understood as the basis for the specific motivational style of delay aversion (Solanto et al., 2001; Sonuga-Barke, 2002; Sonuga-Barke et al., 1992b), which is seen in some conceptualizations as involved in one of several causal pathways from genes to ADHD. Delay aversion provides an alternative or complementary hypothesis to the so far dominant inhibition models of ADHD (Barkley, 1997; Oosterlaan et al., 1998; Schachar and Logan, 1990). It is most often measured by tasks which give a choice between small immediate rewards and large delayed rewards, sometimes presented in a graduating paradigm (de Wit and Richards, 2004; Green et al., 1994; Rapport et al., 1986; Sagvolden et al., 1998; Schweitzer and Sulzer-Azaroff, 1995; Solanto et al., 2001; Sonuga-Barke et al., 1998, 1992b). In all these investigations the person has to weigh up the amount of reward against the level of delay. A critical measure is the inversion point, at which the amount of reward is not sufficient any more to compensate for the increase in delay. ADHD children tend to prefer small, immediate rewards over large, delayed rewards to a greater extent than controls do (Barkley et al., 2001; Kuntsi et al., 2001; Solanto et al., 2001; Sonuga-Barke et al., 1992b). This behaviour can be interpreted as an increased delay aversion or a reduced sensitivity to rewards.

The task presented in this paper, the Continuous Delay Aversion Test (ConDAT) has been developed as a new index for the shortened delay gradient endophenotype. It is based on the same concept as most tasks of delay aversion or reward discounting (Crean et al., 2000; Mischel, 1974; Rapport et al., 1986; Sonuga-Barke et al., 1992b; Sugiwaka and Okouchi, 2004) and aims to measure a tendency towards choosing a small immediate reward instead of a large delayed reward by the means of a multi-trial decision task which produces changing reward delays dependent on different reward sizes. However, it differs from previous methods as it is the first task which is able to monitor delay aversion online so as to provide a continuous output measure representing delay tolerance, while existing tasks often are limited to a certain age range or diagnostic category and therefore produce ceiling effects (International Multi-Centre ADHD Genetics Project (IMAGE), unpublished data). The ConDAT overcomes these problems by means of an unlimited trial duration, which deals with a continuously chang-

ing reward/delay ratio in order to continuously increase the sensitivity to delay aversion during each trial. While waiting is strongly rewarded at the beginning of a trial, it becomes more and more unattractive during a trial, because the increment of reward per time decreases and tends towards zero. The total time a person invests for collecting rewards does not only influence the amount of the (real) reward, but also leads to a delay of the (real) reward, which is given immediately at the end or the task (see Fig. 1). Additionally, the ConDAT is applicable in neurophysiological experiments, e.g. recording of event-related potentials, or in experiments using brain imaging techniques, e.g. functional magnetic resonance imaging. Therefore, the task is written in the language of Presentation(r) Software (Version 8.80; for detailed description see http://nbs.neuro-bs.com), which allows an exact and comprehensive control over stimuli and responses as well as code delivery to external recording devices. This study aims to collect pilot data for the ConDAT from children and give evidence for construct validity and test–retest reliability by comparing the data to behavioural ADHD measures and measuring correlations between test and retest data.

2.3 Material and methods

2.3.1 Description of the task

The ConDAT consists of an online instruction, followed by a short practise trial, 30 main trials with intermittent feedback trials, and a final reward display. The test subject attempts to maximise the amount of time of a preferred video clip by accumulating “gold” over a 30 trial session. The “gold” is accumulated and finally transformed into the duration of the “real reward”, namely the video sequence finally displayed.

The online instruction introduces the proband to the main idea of the task: The relation between video duration, amount of “gold” and delay needed to collect “gold” is explained at the start of the session. The instruction also makes the proband familiar with the story of the task: You have to collect gold from each of 30 “gold-donkeys”, which let out gold from their mouth, as in the fairy tale. The more gold you collect, the more of the video clip you can see. If you collect all the gold from the whole herd, you can see 120 s of the chosen clip. The donkeys only have a certain amount of gold to give.

The gold is flowing very fast at the beginning, but soon the flow diminishes and finally the donkey is completely dried out and has no more gold to give. Because donkeys are stubborn, from time to time the donkeys stop letting out the gold, although they will still have some in their stomach, indicated by the stopping of the flow of gold and the appearance of a red question mark. In this case, you can wheedle more gold out of the donkey by pressing this button. If you don't want to wait any longer for gold of a certain donkey, you can call the next donkey by pressing this button here. The instruction also explains the feedback, which informs the proband about the remaining trials and about the estimated video duration. The instructing person has to be sure about the probands correct understanding of the task and should repeat instructions, or practice trial, if necessary. At the end of the instruction a screenshot and the title of each of the four video clips are presented and the program is linked to the video clip chosen.

In each main trial, the reward accumulation is visualised as a bucket, which is filled up through a gold flow from the donkey's mouth (see Fig. 2). The width of the jet of gold coming out of the donkey's mouth is diminished according to the calculated flow and then replaced by single drops, which fall more and more seldomly and finally stop.

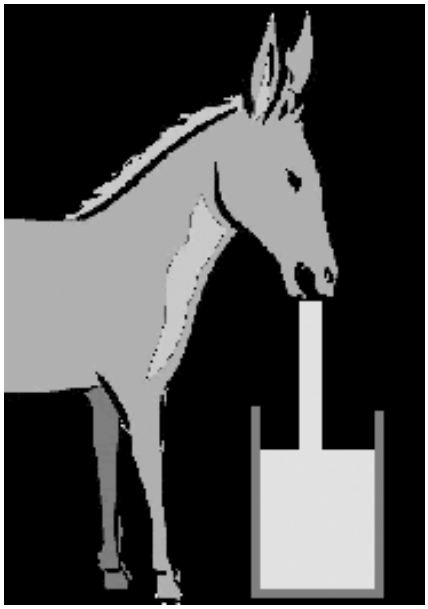

Figure 2. Visualisation of reward accumulation in a ConDAT trial.

The slope reflecting the accumulation of "gold" per unit of time (see Fig. 3a) is very steep at the beginning and flattens out very soon after the beginning of a trial: only a mean net waiting time of 45 frames (4.5 s = 7.5% of the rewarded trial duration) in each of the 30 trials is necessary for seeing 95% (114 of 120 s) of the video clip; 175 frames (17.5 s = 29%) for seeing 99% (119 s). In other words, the last second of the video has to be paid by 70% of the maximal waiting time (42.5 s) in each trial. Beyond the 600th frame waiting is not rewarded any more (but still registered by the program) and therefore becomes fully ineffective. The logarithmic function of the current version of the CoNDAT has been tuned in sev-

eral preliminary versions of the task in order to provide a measure which is able to differentiate between individuals within an unlimited age-range and/or across a wide psychopathological spectrum.

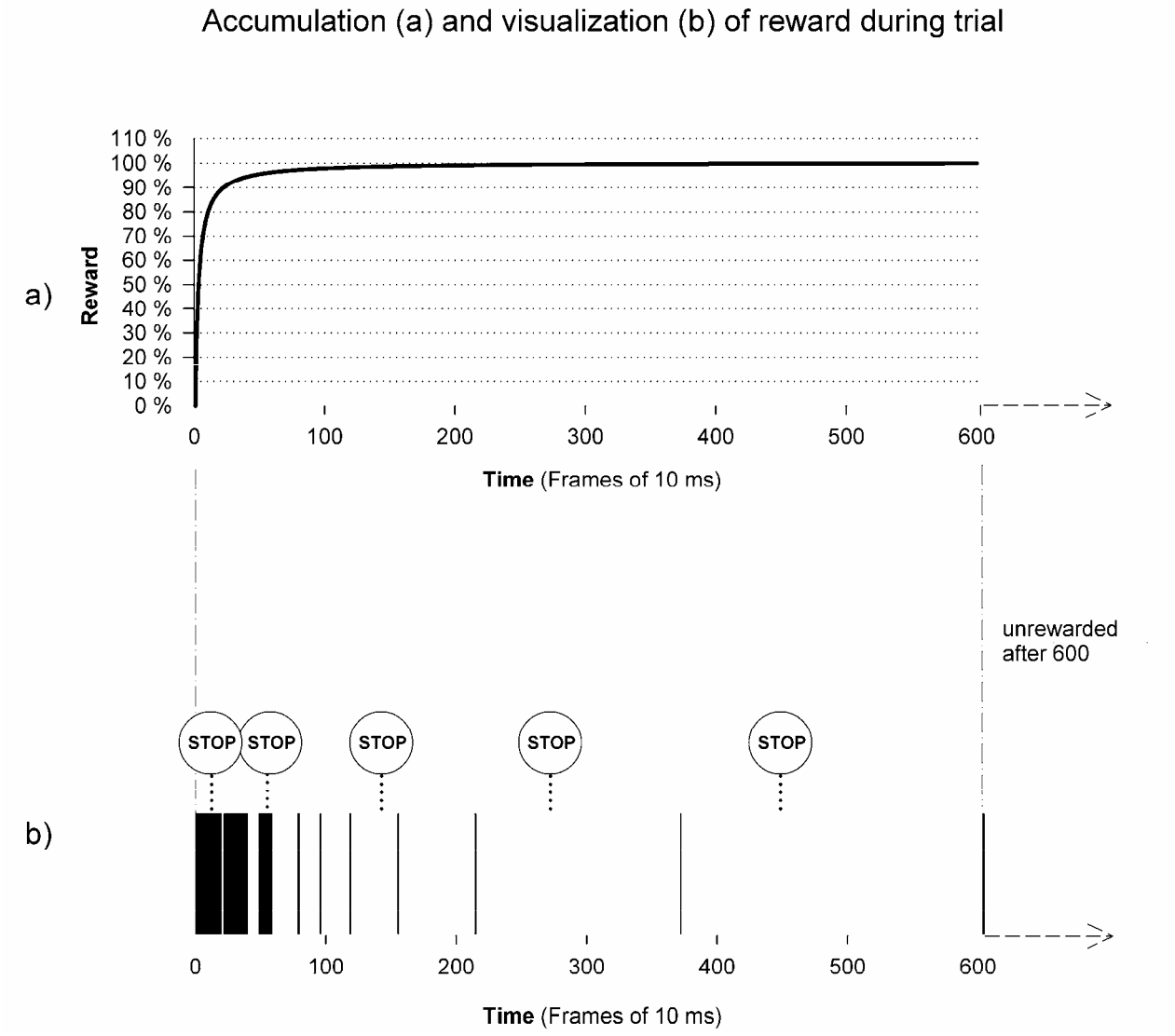

Figure 3. (a) Reward accumulation. The accumulation of the reward follows a logarithmic function of the elapsed time. (b) Visualisation of reward accumulation. During the black marked time periods the reward accumulation is visualised as an increasing gold level of the container. During the white marked periods no reward accumulation is visible. At the five time points indicated by the STOP-signals the reward accumulation stops and proceeds only at the proband's keystroke. Dropping a trial and calling the next trial by another keystroke is possible at any time.

The extremely inefficient reward conditions associated with waiting during the later part of the trial is necessary in order to be sensitive also for “reward maximisers”, i.e. people who are willing to spend much time for little reward. Because not only the length but also the start time delay of the video is dependent on the invested time for collection, waiting is directly linked not only to the amount but also to the delay of reward. Delay aversion should therefore lead to early trial termination in order to avoid delay.

As an additional feature, the filling of the bucket stops five times within each trial (after 1.1, 5.5, 14.2, 27.3 and 44.7 s) and must be reactivated by a participant keystroke (see Fig. 3b). On one hand, the attention of the proband is kept on task by that feature. On the other hand, these two functionally different responses (a “go-on”-response and a drop-response) can be investigated in terms of corresponding event related EEGpotentials (Brandeis et al., 1998; Steger et al., 2000) which allow the comparison of an aversive neuronal state (drop-response) to an affirmative volitional neuronal state (“go-on”-response). Additionally, due to the comprehensive protocol, unnecessary mouse clicks, which occur often in hyperactive children, are logged and can be interpreted as a secondary hyperactivity measure. The feedback trial, which is displayed after each main trial, shows both visual bars and verbal information about the remaining trials and the projected video duration based on the mean duration of all trials completed so far. After the last feedback trial, a waiting period of 120s minus the calculated video duration is inserted, before displaying the video clip. Thus, the reward delivery ends exactly 120s after the end of the last feedback trial, independent of the amount of the video-reward.

2.3.2 Outcome measures

The main outcome measure is the delay interval that the participants are prepared to wait before terminating each trial. This is displayed as the percentage of maximal rewarded trial frames, i.e. of 30x600 frames. The calculation of the video duration is based on this frames-based waiting duration. One hundred twenty seconds of a video are displayed, if a proband never presses the drop button before the 600th frame. Additional waiting beyond the 600th frame and during the filling pauses is not rewarded.

The final result file also displays the mean additional, not rewarded waiting time and the mean total waiting time. The logfile includes a detailed time protocol of all displayed frames, all stimuli and responses at a resolution of 1 ms. From that logfile, additional measures can be derived, such as unnecessary mouse-clicks, mean trial duration, regression of trial duration over task. If the task is used in a neurophysiological or imaging design, every stimulus and response event as well as all major changes of screen display can be sent as specific programmable codes to a recording device.

2.3.3 Technical requirements

The ConDAT task requires Presentation(r) Software Version 0.80 or higher, installed on aWindows based computer with good video and sound capacities (for exact technical specifications see http://nbs.neuro-bs.com). If a notebook computer is used, external loudspeakers are recommended.

2.4 Preliminary results

A pilot study using a regular first grade school class of 7 boys and 11 girls with a mean age of 84.9 months (S.D. = 5.5) is reported here that explores aspects of ConDAT. The teacher completed the Strength and Difficulties Questionnaire (SDQ; Goodman, 1997) and rated each child's global ambition and global endurance. Both global measures were introduced after ConDAT results in a sibling-study of ADHD-children gave evidence that additional, and probably more personality-related, factors be side ADHD-psychopathology influence motivational behaviour (unpublished data). The teacher had to rate whether the child "in general aims high" (global ambition) and whether the child "in general can pursue an aim over a longer period" (global endurance) on a five-point Likert-scale (1 = not at all, 2 = a little, 3 = moderate, 4 = clearly, 5 = absolutely true). For reasons of reliability this rating was repeated after 12 weeks, when the task was administered a second time. The correlation between the first and the second measurement was $r = 0.685$ ($p = 0.002$) for global ambition and $r = 0.692$, p (0.001) for global endurance. The mean trial duration of theConDATwas log transformed in order to normalize

the data. The log transformed mean trial duration correlated significantly (r = 0.59**, p = 0.010) with the same measure after 12 weeks. The trial duration (see Fig. 4) of the sample diminished from about 25 s (42% of rewarded trial length) at the beginning to about 15 s (25% of rewarded trial length) at the end of the session. The mean trial duration of the sample was 19.9 s (S.D. = 9.4 s, min = 10.5 s, max = 50.1 s) or 33% of the trial length. This shortening of the trial time during the session was measured by regression analysis: the mean slope of the trial time (linear regression coefficient B) was −3.58 (S.D. = 4.5, min =−13.2, max = 3.9) and differed significantly from the slope of a constant trial time (=0) over session (one sample t-test: t = 3.45, p < 0.003). The log transformed mean trial durations were negatively correlated withSDQhyperactivity scores and positively with global endurance, but not correlated with other behavioural measures (Table 1).

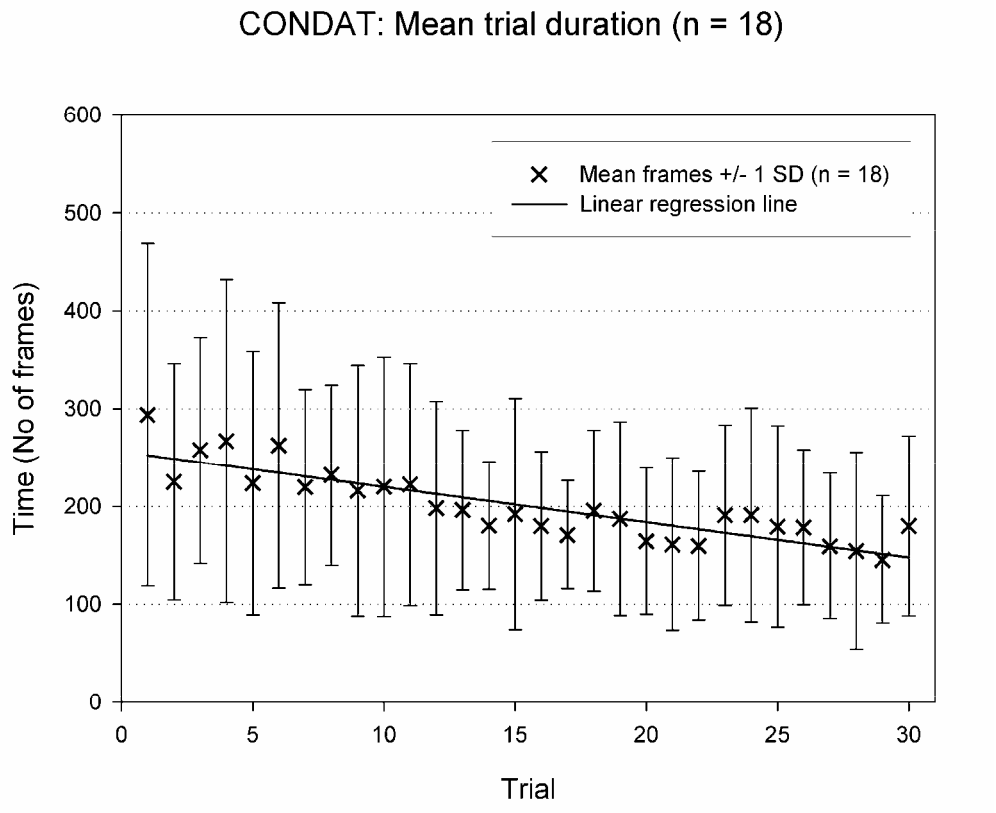

Figure 4. Course of the mean trial duration (frames of 1/10 s) of the whole sample (n = 18). Each trial only ends by the proband's intervention. Time periods without reward accumulation, i.e. between the trial stop signals and the go-response and after the 600th frame, are not included in the calculation of the trial duration

			Mean trial duration (log transformed)	
n = 18	Mean	SD	Pearson Correlation	Sig. (2-tailed)
SDQ Total score	13.22	7.092	-.351	.150
SDQ Emotional symptoms	2.00	2.401	.016	.951
SDQ Conduct Problems	2.67	2.114	-.338	.170
SDQ Hyperactivity / Inattention	4.28	3.102	-.470*	.049
SDQ Peer relationship problems	4.17	2.036	-.157	.534
SDQ Pro-social behaviour	4.22	1.629	.209	.405
Global endurance	3.22	1.517	.472*	.048
Global ambition	2.89	1.278	-.053	.834

Table 1: sBehavioural measures and their correlation to ConDAT performance (mean trial duration)

**Significant correlation ($p < .05$).*

2.5 Discussion

The task presented in this paper was developed as a novel measure for delay aversion, a key ADHD-endophenotype. Compared to existing tasks the ConDAT is able to monitor delay aversion online and measure the critical temporal threshold, i.e. the moment when the amount of reward is not sufficient any more to compensate for waiting. The logarithmic and asymptotic reward/delay ratio prevents floor or ceiling effects and provides a sensitivity which should not be limited by age or psychopathological status. Possible biases due to different age-, sex- or income-states are minimised by implementing a reward system that allows the choice of a subjectively attractive reward. The interval scaled output measure not only allows interindividual comparisons at high resolution, but also calculation of intra-session time course, e.g. regression of trial duration, as shown in the presented data.

The decrease of trial duration during task is interpreted as an increase of delay aversion over time or decrease of sensitivity to/interest in the reward. It additionally can be influenced by a explicit or implicit realization, that the later a trial occurs, the less it can alter the mean of the collected symbolic

rewards, which is the basis of the final real reward. The usefulness of the additional features (e.g., protocol of unnecessary mouse clicks, online codes to external recording device) is not demonstrated so far, but show the potential of the task for further neuropsychological investigations and particularly for EEG-recording. The test–retest reliability of the task is adequate, especially when one takes into account the large time interval of 12 weeks and the relatively complex and unstable nature of the measured motivational construct. The significant correlation between the mean trial time and the SDQ hyperactivity score, but not with other SDQ scores, can be seen as a sign of external validity in relation to ADHD symptoms.

As the findings reported here are based on a relatively small sample size and a restricted age range, future research should include more subjects in a wider age range. The study would have benefited from access to parent ratings to test the issue of pervasiveness. Direct comparisons of children with the diagnosis ADHD with healthy controls will give a more valid information about the power of the task in differentiating between these two groups.

Nevertheless, the presented data give evidence for the Con-DAT as a useful addition to the battery of tasks used to measure delay aversion. Compared to existing tasks, it allows a more direct and precise recording of delay aversion related processes due to its online characteristics and its asymptotic open-end trial course. Taken together with the interface for neurophysiological and imaging experiments, it seems to be an instrument that can provide endophenotypical data of a quality that is demanded by Castellanos and Tannock: continuously quantifiable, able to predict disorder, closer to causing agents than diagnostic categories and neuroscientific based.

2.6 References

Almasy L, Blangero J. Endophenotypes as quantitative risk factors for psychiatric disease: rationale and study design. Am J Med Genet 2001;105: 42–4.

Barkley RA. Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. Psychol Bull 1997;121: 65–94.

Barkley RA. Attention deficit hyperactivity disorder: a handbook for diagnosis and treatment. New York: Guilford Press; 1998.

Barkley RA, Edwards G, Laneri M, Fletcher K, Metevia L. Executive functioning, temporal discounting, and sense of time in adolescents with attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). J Abnorm Child Psychol 2001;29:541–56.

Barkley RA, Koplowitz S, Anderson T, McMurray MB. Sense of time in children with ADHD: effects of duration, distraction, and stimulant medication. J Int Neuropsychol Soc 1997;3:359–69.

Barnett R, Maruff P, Vance A, Luk ES, Costin J, Wood C, et al. Abnormal executive function in attention deficit hyperactivity disorder: the effect of stimulant medication and age on spatial working memory. Psychol Med 2001;31:1107–15.

Biederman J, Milberger S, Faraone SV, Kiely K, Guite J, Mick E, et al. Family-environment risk factors for attention-deficit hyperactivity disorder. A test of Rutter's indicators of adversity. Arch Gen Psychiatry 1995;52:464–70.

Bradley JD, Golden CJ. Biological contributions to the presentation and understanding of attention-deficit/hyperactivity disorder: a review. Clin Psychol Rev 2001;21:907–29.

Brandeis D, van Leeuwen TH, Rubia K, Vitacco D, Steger J, Pascual-Marqui RD, et al. Neuroelectric mapping reveals precursor of stop failures in children with attention deficits. Behav Brain Res 1998;94:111–25.

Brown RT, Freeman WS, Perrin JM, Stein MT, Amler RW, Feldman HM, et al. Prevalence and assessment of attention-deficit/hyperactivity disorder in primary care settings. Pediatrics 2001;107:E43.

Castellanos FX. Toward a pathophysiology of attention-deficit/hyperactivity disorder. Clin Pediatr 1997;36:381–93.

Castellanos FX, Tannock R. Neuroscience of attention-deficit/hyperactivity disorder: the search for endophenotypes. Nat Rev Neurosci 2002;3: 617–28.

Crean JP, de Wit H, Richards JB. Reward discounting as a measure of impulsive behavior in a psychiatric outpatient population. Exp Clin Psychopharmacol 2000;8:155–62.

de Wit H, Richards JB. Dual determinants of drug use in humans: reward and impulsivity. Nebr Symp Motiv 2004;50:19–55.

Faraone SV, Perlis RH, Doyle AE, Smoller JW, Goralnick JJ, Holmgren MA, et al. Molecular genetics of attention-deficit/hyperactivity disorder. Biol Psychiatry 2005;57:1313–23.

Goodman R. The strengths and difficulties questionnaire: a research note. J Child Psychol Psychiatry 1997;38:581–6.

Gottesman II, Gould TD. The endophenotype concept in psychiatry: etymology and strategic intentions. Am J Psychiatry 2003;160:636–45.

Green L, Fry A, Myerson J. Discounting of delayed rewards: a life-span comparison. Psychol Sci 1994;5:33–6.

Kent L. Recent advances in the genetics of attention deficit hyperactivity disorder. Curr Psychiatry Rep 2004;6:143–8.

Kuntsi J, Oosterlaan J, Stevenson J. Psychological mechanisms in hyperactivity: I. Response inhibition deficit, working memory impairment, delay aversion, or something else? J Child Psychol Psychiatry Allied Disciplines 2001;42:199–210.

Milberger S, Biederman J, Faraone SV, Guite J, Tsuang MT. Pregnancy, delivery and infancy complications and attention deficit hyperactivity disorder: issues of gene–environment interaction. Biol Psychiatry 1997;41:65–75.

Mischel W. Processes in delay of gratification. In: Berkowitz L, editor. Advances in experimental child psychology. New York: Academic Press; 1974. p. 239–92.

Nigg JT. Is ADHD a disinhibitory disorder? Psychol Bull 2001;127:571–98.

Oosterlaan J, Logan GD, Sergeant JA. Response inhibition in AD/HD, CD, comorbid AD/HD + CD, anxious, and control children: a meta-analysis of studies with the stop task. J Child Psychol Psychiatry Allied Disciplines 1998;39:411–25.

Oosterlaan J, Sergeant JA. Response inhibition and response re-engagement in attention-deficit/hyperactivity disorder, disruptive, anxious and normal children. Behav Brain Res 1998;94:33–43.

Paule MG, Rowland AS, Ferguson SA, Chelonis JJ, Tannock R, Swanson JM, et al. Attention deficit/hyperactivity disorder: characteristics, interventions and models. Neurotoxicol Teratol 2000;22:631–51.

Rapport MD, Tucker SB, DuPaul GJ, Merlo M, Stoner G. Hyperactivity and frustration: the influence of control over and size of rewards in delaying gratification. J Abnorm Child Psychol 1986; 14:191–204.

Sagvolden T, Aase H, Zeiner P, Berger D. Altered reinforcement mechanisms in attention-deficit/ hyperactivity disorder. Behav Brain Res 1998;94:61–71.

Schachar R, Logan GD. Impulsivity and inhibitory control in normal development and childhood psychopathology. Dev Psychol 1990;26:710–20.

Schweitzer JB, Sulzer-Azaroff B. Self-control in boys with attention deficit hyperactivity disorder: effects of added stimulation and time. J Child Psychol Psychiatry Allied Disciplines 1995; 36:671–86.

Sergeant J. The cognitive-energetic model: an empirical approach to attentiondeficit hyperactivity disorder. Neurosci Biobehav Rev 2000;24:7–12.

Shastry BS. Molecular genetics of attention-deficit hyperactivity disorder (ADHD): an update. Neurochem Int 2004;44:469–74.

Smith A, Taylor E, Rogers JW, Newman S, Rubia K. Evidence for a pure time perception deficit in children with ADHD. J Child Psychol Psychiatry Allied Disciplines 2002;43:529–42.

Solanto MV, Abikoff H, Sonuga-Barke E, Schachar R, Logan GD, Wigal T, et al. The ecological validity of delay aversion and response inhibition as measures of impulsivity in AD/HD: a supplement to the NIMH multimodal treatment study of AD/HD. J Abnorm Child Psychol 2001;29:215–28.

Sonuga-Barke EJ. Psychological heterogeneity in AD/HD—a dual pathway model of behaviour and cognition. Behav Brain Res 2002;130:29–36.

Sonuga-Barke EJ, Saxton T, Hall M. The role of interval underestimation in hyperactive children's failure to suppress responses over time. Behav Brain Res 1998;94:45–50.

Sonuga-Barke EJ, Taylor E, Heptinstall E. Hyperactivity and delay aversion—II. The effect of self versus externally imposed stimulus presentation periods on memory. J Child Psychol Psychiatry Allied Disciplines 1992a;33:399–409.

Sonuga-Barke EJ, Taylor E, Sembi S, Smith J. Hyperactivity and delay aversion—I. The effect of delay on choice. J Child Psychol Psychiatry Allied Disciplines 1992b;33:387–98.

Steger J, Imhof K, Steinhausen H, Brandeis D. Brain mapping of bilateral interactions in attention deficit hyperactivity disorder and control boys. Clin Neurophysiol 2000;111:1141–56.

Sugiwaka H, Okouchi H. Reformative self-control and discounting of reward value by delay or effort. Jpn Psychol Res 2004;46:1–9.

Swanson J, Castellanos FX, Murias M, LaHoste G, Kennedy J. Cognitive neuroscience of attention deficit hyperactivity disorder and hyperkinetic disorder. Curr Opin Neurobiol 1998;8:263–71.

Taylor E. Clinical foundations of hyperactivity research. Behav Brain Res 1998;94:11–24.

Wilens TE, Biederman J, Spencer TJ. Attention deficit/hyperactivity disorder across the lifespan. Annu Rev Med 2002;53:113–31.

3 Studie 2: Willingness to Wait for Rewards in Childhood and Adolescence in the Face of Diminishing Returns [1]

3.1 Abstract

A reward becomes less attractive, the more delayed it is. Previous studies suggest this tendency is most pronounced with young children and declines as children grow. Here age-related change of delay tolerance is investigated with a new adaptive computer task, the Continuous Delay Aversion Test (ConDAT) designed to schedule diminishing reward return with growing delay in order to differentiate delay sensitivity across a wide age and delay-tolerance range. 183 children between ages 6 to 17 years were presented with the ConDAT and two components were studied: delay tolerance in the face of diminishing returns on each trial and changes in delay tolerance across trials in a session. Results show that delay tolerance increases with age and decreases with time on task. The effect of time-on-task is most pronounced in children and for individuals with high hyperactivity symptoms. Our findings give evidence for a dynamic and complex concept of the development of tolerance to delayed reward which interacts with aspects of sustained performance over time. The ConDAT successfully overcame several constraints of standard choice delay tasks. It appears to be especially suited for developmental studies.

3.2 Introduction

Typically our ability to choose long-term goals when faced with a short-term gratification reduces the further that goal is the future. This phenomenon is called temporal discounting and is found in many

[1] Müller, U. C., Sonuga-Barke, E., & Steinhausen, H. C. (2009). Manuscript submitted to Developmental Psychology.

animal species and also in humans (Mischel & Ayduk, 2004). In the 1970s, temporal discounting was extensively investigated in young children by using the delay of gratification paradigm. This offers the choice between a smaller immediate reward, (e.g. one cookie), and a delayed larger reward, (e.g. two cookies; Mischel & Baker, 1975). This simple task when applied to young children was a powerful predictor of academic and social competence in adolescence (Mischel, Shoda, & Peake, 1988). The choice of either the immediate small reward or the delayed large reward is assumed to be an indicator of the amount of self-regulatory competences necessary to withstand immediate gratification in order to access long term rewards (Mischel & Ayduk, 2004). Given that these regulatory abilities develop rapidly during childhood it is not surprising that tolerance for delay before rewards increases with age (Bjork, et al., 2004; Green, Fry, & Myerson, 1994; Walls, 1973). It seems more developed in females (Silverman, 2003) while failures of delay of gratification are associated with emergence of substance abuse (Petry, 2002; Reynolds, Richards, Horn, & Karraker, 2004) and attention-deficit/hyperactivity disorder (ADHD; Marco, et al., 2008; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986; T. Sagvolden, Aase, Zeiner, & Berger, 1998; Sonuga-Barke, Taylor, Sembi, & Smith, 1992), which has been explicitly denoted as a disease of self regulation (Barkley, 1998).

Studies of the psychopathophysiology of these disorders have been a strong driving force in the development of understanding of the mechanism involved in delay tolerance (e.g. T. Sagvolden, et al., 1998; Sonuga-Barke, 2005). Findings from ADHD may be especially relevant to normal psychology, as it is increasingly understood as a quantitative trait, which is continuously distributed in the population, and the risk factors for which overlap with factors influencing traits in the normal population (Asherson & Image Consortium, 2004). Recent evidence against the conception of ADHD as a distinct disorder is provided by a study of brain development in ADHD, which has shown that the cortical structures associated with attention differed between controls and children with ADHD, but the development trajectories are the same in both groups (Shaw, et al., 2006). Indeed the notion of delay tolerance is central to recent models of ADHD (Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). For instance the delay aversion hypothesis argues that ADHD children's symptoms are underpinned by a motivation to escape or avoid delay (Sonuga-Barke, et al., 1992) which together with an impulsive

drive for immediate reward (Terje Sagvolden, Johansen, Aase, & Russell, 2005) creates a strong preference for small immediate over large delayed rewards.

Studies of abnormal populations have also driven innovations in measurement to some degree. So for instance, the Continuous Delay Aversion Test (ConDAT; Müller, Sonuga-Barke, Brandeis, & Steinhausen, 2006) was designed to overcome several problems in measuring delay tolerance, from which the most important one is a limited range of sensitivity leading to ceiling or floor effects (e.g. Marco, et al., 2008) found in ADHD populations raining widely in age. Standard tasks developed for children are too easy for adolescents and tasks developed for adolescents are too difficult for children. These limitations have to do with developmental factors, e.g. an increasing tolerance for delay with age (Green, et al., 1994; Scheres, et al., 2006) and the diminishing salience of material rewards, particularly monetary rewards, with age (Walls, 1973).

The ConDAT, similar to other delay aversion tasks which force the choice between sooner smaller and later larger rewards and measure the tendency towards one reward type, e.g. the Maudsley Index of Delay Aversion (MIDA; Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001), the ConDAT positively associates the amount of the reward with its delay. However, instead of offering the choice between two fixed choice options at the beginning of each trial, the task continuously delivers a stream of token rewards and participants are asked to say at which point they want to stop the trial and get the accumulated reward (see Figure 1). Crucially each trial incorporates a diminishing returns scenario in which the delivery rate of rewards gets less as the trial progresses. The rate is far higher in the beginning than at the end of each trial. Thus, this reduction in value for each time unit should heighten the sensitivity to delay especially towards the end of the trial so that even the most delay tolerant individual would be challenged to show some delay intolerance (A more detailed description and sample data will be provided in the methods section and in the appendices).

Therefore the main aim of the present study was to investigate the development of tolerance for delayed reward delivery under diminishing returns in individuals across children and adolescents. A

second aim was to examine the extent to which tolerance for delay persists over a session by studying performance as a function of time-on-task. On the one hand such a measure is of importance with respect to a possible novelty effect, which can be present particularly at the beginning of the task and probably reduces the aversive character of the situation (Antrop, Roeyer, Oost, & Buysse, 2000). On the other a lack of effortful persistence on demanding tasks is hallmark of the performance of young children (Lin, Hsiao, & Chen, 1999) and also children with psychopathology (Seidel & Joschko, 1990). Finally, we were interested in examining the impact of individual differences in IQ, gender and psychopathological measures on delay aversion using ConDAT. This allowed us to compare the associates of performance of the task used in the current study with those found in previous studies.

In general in the line with the general notion of increasing capacity for impulse control with age we predicted that delay tolerance would increase with age. We also predicted it would be higher in females (Silverman, 2003) and lower in children with psychopathology (Marco, et al., 2008; Müller, Schönenberger, Sonuga-Barke, & Steinhausen, 2009). Based on findings of Marco et al. (2009) one may further expect that time spent on task is positively correlated with IQ. However, we must also be aware that the salience of the reward (viewing a video clip) is likely to decrease with age and this might reduce the preference for delay with age. This latter effect may be ameliaorated by giving the participants the possibility of viewing a clip of one's own choice.

In terms of time on task we predicted that in general delay tolerance would decline across session and we predicted that this failure of persistence would be more marked with young compared to older participants. As observed in pre-studies, younger children often are enthusiastic and ambitious at the beginning of the task, so that initially there are long waiting times which decrease as the task passes. More stable waiting times across the session are expected to be seen in older children. Moreover, such time-on-task effects on delay choice are found in studies about sustained attention in ADHD (Heinrich, et al., 2001; Seidel & Joschko, 1990) and so we also expected a decline in delay tolerance across sessions to be related to ADHD symptoms.

3.3 Methods

3.3.1 Participants

Children were recruited from schools in the German speaking part of Switzerland. A total of 191 children from nine school classes with grades from 1 to 9 participated in the study. Exclusion criteria were IQ<70, a diagnosis of schizophrenia or autism, any neurological disorders of the central nervous system, or a genetic disorders that might mimic ADHD. After excluding four children with missing ConDAT data, 3 children with an IQ < 70, and 1 child with an ADHD diagnosis, the final sample consisted of 90 boys and 93 girls between age 6 and 17 (see Table 1). The age of the sample follows almost a normal distribution (Kolmogorov-Smirnov-Z = .973, p = .305). There was no sex difference between school classes (see Table 1).

3.3.2 Measures

Delay Tolerance Measure

The basic idea of the ConDAT is described in the introduction and a detailed description of the task is provided in the appendix. The task was programmed in the code of the Presentation® Software (see http://nbs.neuro-bs.com) and runs on a conventional computer with Windows® OS 98 and higher. The computer program includes all parts of the task, i.e. instruction, three practice trials, 30 main trials, and the rewarding video clip.

The most important primary output parameter is the rewarded waiting time in each trial, representing the amount of delay a person is willing to accept with regard to the corresponding reward. The reward size is based on all periods of the session which actually are accessible for collecting the token from the subject's view. Consequently, the 30 feedback trials and the decision periods, i.e., the time a subject needs to consider and perform a response to the programmed repeated freezing of the rewarding process (see Figure 1) are not rewarded. The sample in Appendix II demonstrates the relation between the actual time-on-task and the rewarded time.

Table 1

Demographic findings

School class	A	B	C	D	E	F	G	H	I	All	*Class effects* χ^2	*p*
Age: mean (SD)	7.0 (0.48)	8.1 (0.43)	9.4 (0.51)	11.1 (0.55)	11.5 (0.44)	12.2 (0.27)	13.6 (0.63)	14.4 (0.46)	15.9 (0.62)	11.3 (2.70)		
Age: min - max	6.1 - 8.2	7.5 - 9.4	8.6 - 10.4	10.2 - 12.0	10.9 - 12.5	11.5 - 12.7	12.6 - 15.1	13.5 - 15.3	14.9 - 16.9	6.1 - 16.9		
School: grade / type	1 / P	2 / P	3 / P	5 / P	5 / P	6 / P	7 / S	8 / S	9 / S			
N: Total (males)	17 (6)	23 (13)	26 (15)	19 (12)	20 (9)	20 (11)	21 (9)	21 (9)	16 (6)	183 (90)	5.79	0.651

Note. P = Primary school; S = Secondary school

Four main measures are derived from this basic variable:

- The overall mean waiting time (in seconds per trial) as a measure of delay tolerance (DT). Because this measure was skewed it was log transformed (DTlog) prior to analysis. For some analyses this measure was broken down into five sections (Trials 1-7; 8-14; 15-21; 22-28; 29 and 30), which were named DT1 to DT5, and DTlog1 to DTlog5.
- The slope of the linear regression line (Beta coefficient; in seconds per trial) calculated across the waiting times of all trials except the last two trials, i.e. from trial 1 to trial 28 as measure of the persistence of delay tolerance (PERSIST).
- The standard deviation of waiting times across trials (DTVAR).
- A measure of fluctuation or stability of the waiting time, calculated as the mean of the absolute changes between all pairs of adjacent trials (DTFLUC).

Normal distribution was confirmed (following log transformation in the case of DT variables) by the Kolmogorov-Smirnov Test (all $Z < .78$, *all* $p > 0.56$).

Psychopathology Questionnaires

To assess the teachers perception of ADHD symptoms and comorbid problems, the long form of the revised Conner's Teacher Rating Scale, CTRS-R:L (Conners, 1997) and the Strength and Difficulties Questionnaire (Goodman, 1997) were administered. The CTRS-R:L summarizes 59 questions into the following 14 scales: Oppositional Behaviour, Cognitive Problems/Inattention, Hyperactivity, Anxious/Shy, Perfectionism, Social Problems, ADHD Index, Global Index: Emotional, Global Index: Impulsivity, Global Index: Total, DSM–IV ADHD Symptoms: Inattention, DSM–IV ADHD Symptoms: Hyperactivity/Impulsivity, and DSM–IV ADHD Symptoms: Total. The raw scores of the CTRS:R-L were transformed into T-scores using the US standardization sample. In a further step T-scores were transformed into two new categories, namely, "normal" (scores < 60) and "affected" (scores >= 60).

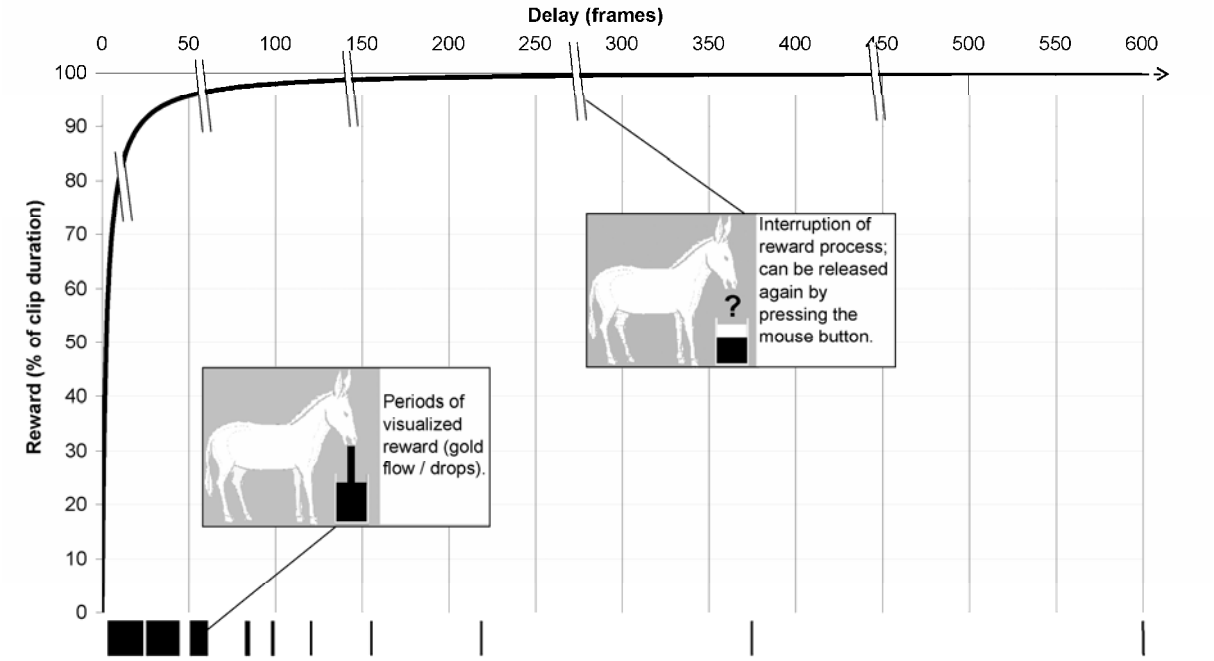

Figure 1 Reward mechanism of the ConDAT. The curve indicates the cumulative amount of returned token during a trial, which is visualized by a steadily diminishing stream of gold out of a donkeys mouth into a container (Black bars at the bottom of the figure indicate the phases of actual gold flow, adapted to the slope of the curve). The token delivery process is completely stopped from time to time (double backslashes) and can be reactivated by pressing a mouse button. Each trial can be terminated at any time by pressing the spacebar.

The SDQ contains 25 questions and the five following scales: Emotional Symptoms, Conduct Problems, Hyperactivity, Peer Problems, and Prosocial Behaviour. The Total Difficulties Score is based on summing all scales except the Prosocial Behaviour Scale. The raw scores are dichotomized into an 'affected' and a 'normal' category by assigning the following scores tot the 'normal' category: 0-11 in the Total Difficulties Score, 0-4 in the Emotional Symptoms Score, 0-2 in the Conduct Problems Score, 0-5 in the Hyperactivity Score, 0-3 in the Peer Problems Score, and 6-10 in the Prosocial Behaviour Score (see www.sdqinfo.com). For the purpose of the present study all scores above these thresholds (Prosocial Behaviour Score: below the threshold) were assigned to the category 'affected'. In addition, the teacher had to rate the child's performance in terms of each global ambition and global endurance using a five-point Likert-scale (from 1=not at all, to 5=absolutely true). Both global ratings

(GR) were transformed into new dichotomous variables encluding the categories, “low” (scores 1-3) and “high” (scores 4 and 5).

IQ

Five subtests of the German version of the WISC-III (Tewes, Rossmann, & Schallberger, 2000), i.e. Vocabulary, Similarities, Picture Completion, Block Design, and Digit Span were assessed. IQ was prorated from the first four subtests using a procedure described by Sattler (1992).

3.3.3 Procedure

The parents of all children gave their informed consent, and children orally agreed in participating in the study. Each child was assessed individually during regular classes but in separate rooms near the classroom in two different sessions, one session for the ConDAT task and one for the IQ test.

3.3.4 Data analysis

All Statistics are calculated using the program SPSS (2008). For normally distributed variables (IQ, age, and all variables of the ConDAT task used in inferential statistics) differences between groups were analyzed by t-tests and ANOVAs; associations between measures were established with Pearson correlations. If variables markedly deviated from normality, as all questionnaire scores did, Spearman’s rank correlations or Kruskal-Wallis-Test were calculated.

To investigate the main effects of task sections and age groups and their interactions on ConDAT variables, GLM repeated measurement analyses are performed. To investigate the association between psychopathological scores and ConDAT scores, binary logistic regressions (forward: conditional) were employed with the dichotomized scores of the questionnaire scales as dependent variables and all ConDAT measures plus age as covariates. These post hoc tests are not performed when preceding *t* tests were not significant (e.g. tests including gender), or when only one ConDAT measure differed

between the categories of the target questionnaire and additionally the dependent score already was adjusted for age (e.g. the four CTRS scores).

3.4 Results

3.4.1 Delay choice task

DT and PERSIST, were uncorrelated (r = 0.063, p = 0.397. DTVAR and DTFLUCT WERE highly correlated (r = 0.81, $p < 0.001$), and also correlated with DT (both r between 0.58 and 0.60, both $p <$ 0.001), but not to PERSIST

Change in DT for the whole sample.

As shown in Table 2, the children invested on average 24.6 (11.98) seconds per trial (DT) in collecting tokens, with a minimum of 5.52 seconds and a maximum of 60 seconds. DT per trial decreased slightly during the task as indicated by a mean slope score of - 0.088, which corresponds to a decrease of 2.5 seconds across the trials 1 to 28. The highest increase of a childs waiting time (PERSIST = 2.28) corresponded to a 64 seconds longer waiting time in the 28^{th} trial, compared to the first trial, and the maximal decrease was 43 seconds from trials 1 to 28 (PERSIST = - 1.53). The average DTVAR was 7.95 seconds and the average absolute change from one trial to the next (DTFLUC) was 6.1 seconds.

If the change of the waiting times across the session is decomposed into four sections of 7 trials and a final section of 2 trials, the non-linear character of the change across time is evident (Table 2). For the total sample DT in each of the first two sections was similar, felt down in the sections 3 and 4, and increased to the highest level in the section 5. The overall difference between the sections was significant (Friedman F(4) = 32.3, $p < 0.001$); each of the sections 1, 2, and 5 differed significantly from both sections 3 and 4 (Wilcoxon-Test; all $p <= 0.01$).

Table 2
Delay aversion findings: scores of the ConDAT measures

	Total sample	**Age group 1**	**Age group 2**	**Age group 3**		
N	183	58	75	50		
Age (mean [SD])	11.3 (2.7)	8.1 (0.93)	11.6 (0.88)	14.7 (0.96)		
Age range	6.1 - 16.9	6.12 - 9.72	9.79 - 13.25	13.33 16.92		
ConDAT measures	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*		
Non-normally distributed measures						
DT [#]	24.5 (12.0)	21.2 (10.2)	24.5 (11.8)	28.6 (13.1)		
DT1 [#]	25.3 (12.9)	23.8 (10.9)	25.0 (12.6)	27.5 (15.2)		
DT2 [#]	25.1 (13.5)	22.4 (126)	25.4 (13.7)	27.7 (13.8)		
DT3 [#]	23.7 (13.3)	19.8 (118)	23.2 (12.8)	29.0 (14.3)		
DT4 [#]	23.6 (12.7)	19.1 (107)	23.5 (12.0)	29.0 (14.0)		
DT5 [#]	26.5 (14.9)	20.4 (133)	27.2 (14.0)	32.4 (15.5)		
Normally distributed measures					*ANOVA F*	*Sig. post-hoc pair differences*
DTlog	3.080 (0.51)	2.947 (0.47)	3.072 (0.53)	3.247 (0.47)	4.948 **	1:3 (**), 2:3 (*)
PERSIST [#]	-0.09 (0.50)	-0.258 (0.49)	-0.10 (0.43)	0.023 (0.54)	4.676 **	1:2 (°), 1:3 (**)
DTVAR [#]	7.95 (4.03)	7.75 (4.02)	7.76 (3.79)	8.46 (4.41)	0.552 (n.s.)	-
DTFLUC [#]	6.16 (3.33)	5.58 (2.86)	6.23 (3.41)	6.73 (3.64)	1.657 (n.s.)	-
DTlog1 [a,b]	3.102 (0.52)	3.076 (0.43)	3.079 (0.55)	3.166 (0.55)	0.553 (n.s.)	-
DTlog2 [c,d]	3.076 (0.55)	2.956 (0.57)	3.085 (0.58)	3.200 (0.50)	2.629 °	1:3 (*)
DTlog3 [a,c,e]	3.006 (0.58)	2.827 (0.56)	2.988 (0.60)	3.241 (0.52)	7.252 ***	1:3 (***), 2:3 (**)
DTlog4 [b,d,f]	3.011 (0.57)	2.798 (0.56)	3.021 (0.56)	3.245 (0.52)	8.964 ***	1:3 (***), 2:3 (**)
DTlog5 [e,f]	3.105 (0.61)	2.822 (0.63)	3.159 (0.57)	3.352 (0.53)	11.911 ***	1:2 (**), 1:3 (***)

Note: *DT* = mean waiting time across all trials; *DT1 to DT5* = mean waiting time in sectors 1 to 5;
[#] in seconds *DTlog1 to DTlog5* = log of the mean waiting times; *PERSIST* = Coefficient (B) of the waiting times regressed on trials 1 to 28; *DTVAR* = standard deviation of the mean waiting times across al trials; *DTFLUC* = mean absolute waiting time difference between two adjacent trials. a(**), b(**), c(**), d(*), e(**), f(***) = significant pairwise differences.
° $p < 0.1$ * $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

The effect of age

Age was positively correlated with DT (r = .20, $p = 0.007$) and PERSIST (r = 0.24, $p = 0.001$), indicating that older subjects were more willing to tolerate delay, and this persisted more during the session. As a consequence, if the waiting times per section are analyzed with regard to age, the correlation between age and the mean waiting time increased from section to section; (section 1 (r = .03, n.s); 2 (r = .15, $p = .048$); 3 (r = .25, $p = .001$); 4 (r = .28, $p < .001$) and 5 (r = .31, $p < .001$)). A repeated meas-

urement analysis with section as within-subjects factor and age as covariate confirmed these findings: the main effects of age (F = 10.695, p = 0.001) and section (F = F = 4.236, p = 0.003) and the interaction effect between age and section (F = 3.258, p = 0.013) were significant.

In order to explore further the interaction between age and time-on-task the sample was divided into three age groups of equal age range: low-age level of the primary school (grades 1 - 3), middle-age level of the primary school (grades 4 - 6), and secondary school (grades 7 – 9) . Figure 2 confirmes that both DT and PERSIST increase with age, and post-hoc tests gave significant DT differences between the age groups 1 and 3 (p = 0.001), and significant PERSIST differences between the age groups 1 and 2 (p = 0.003) and the groups 1 and 3 (p < 0.001). There were no age related effects for DTVAR and DTFLUC.

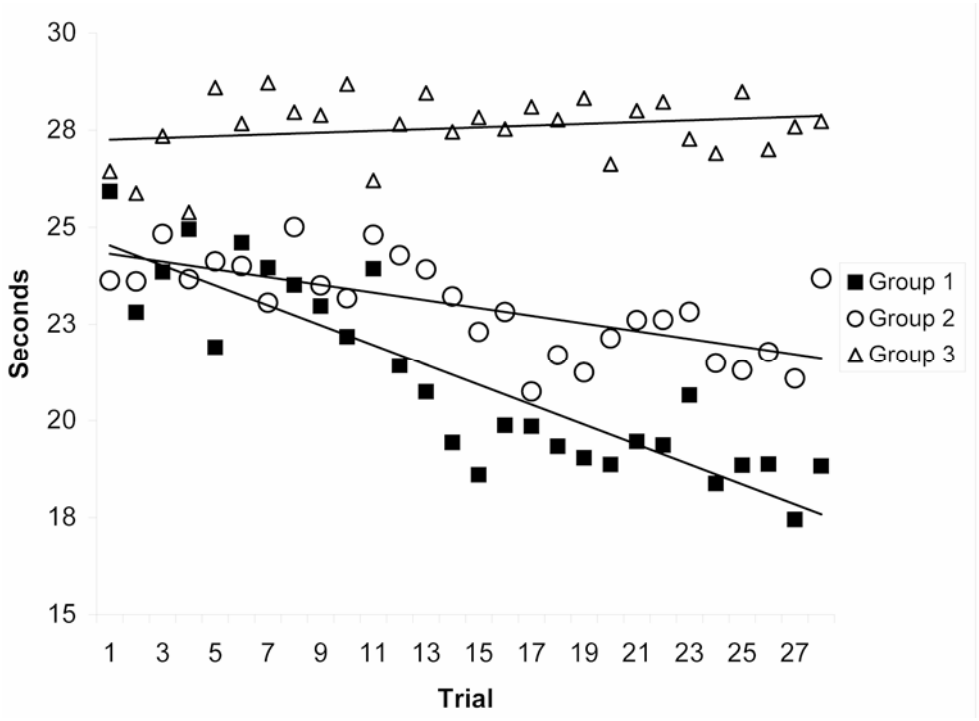

Figure 2. Course of the mean waiting time (DT) in 3 age groups across trials 1-28 of the ConDAT task, including regression lines (PERSIST) in each age group.

Figure 3 shows that these effects are not linear across the session and with age. Very strikingly all groups increased waiting towards the very end, even when the trend has been negative in the previous sections. Further, the age effect seemed to change markedly between sections 2 and 3 (Table 2). When a general linear model with the variables DT1log to DT5log as repeated inter-subjects factors and age group as between subjects factor was performed, there was an univariate effect of age group ($F = 6.486$, $p = 0.002$) and a multivariate effect of section ($F = 7.343$, $p = <0.001$). Pairwise differences between the age groups 1 and 3 were significant ($p = < 0.001$), and between the groups 1 and 2 ($p = 0.051$) and 2 and 3 ($p = 0.056$) close to significant. When sectors are pairwise compared, all differences were significant except between the sectors 3 and 4, and between each pair of the sectors 1, 2, and 5, indicating age related leaps in the middle of the session and close to the end.

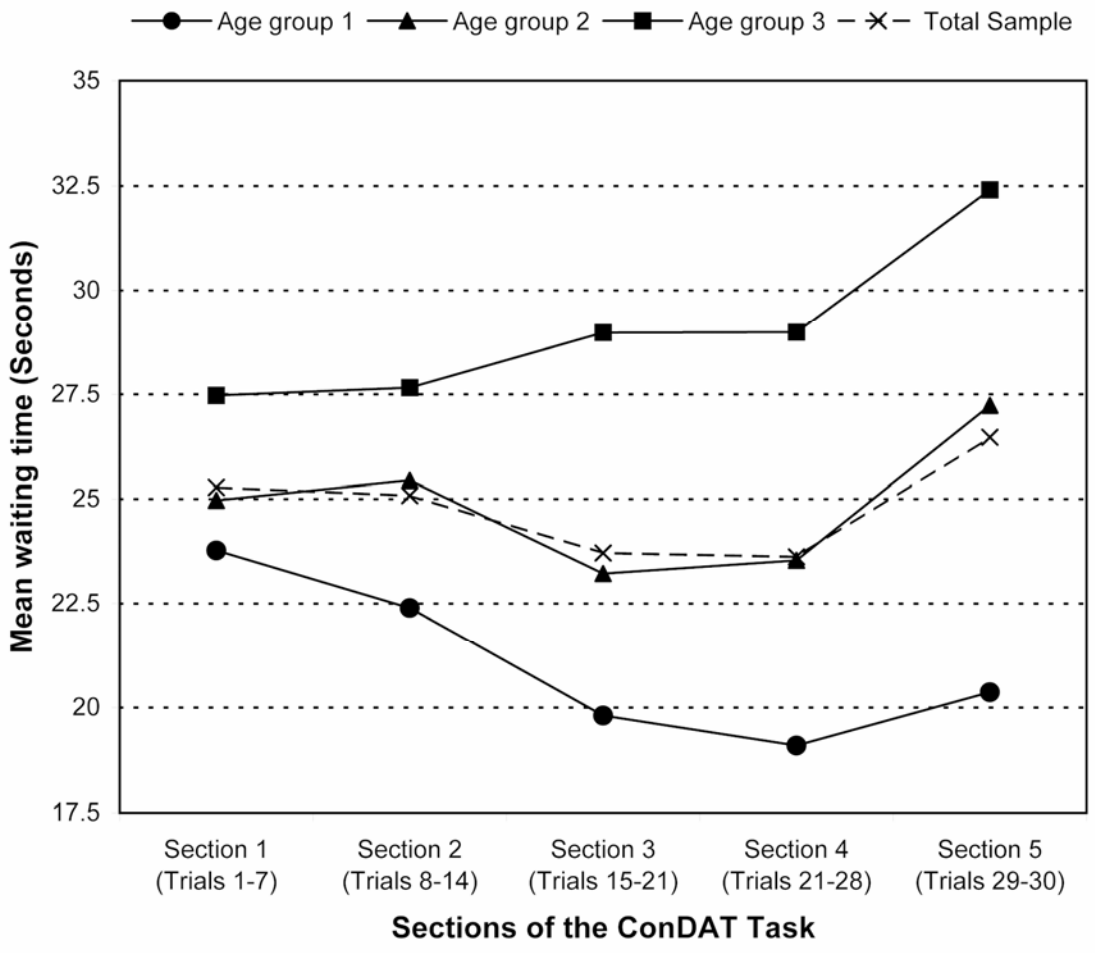

Figure 3. Mean waiting time in the five sections of the ConDAT task by age groups 1 (6-9 years), 2 (9-12 years) and 3 (13-16 years).

Effects of Gender and IQ

There was a trend toward higher DT in females' (see Table 3) but no significant association between this or other ConDAT measures and gender. None of the ConDAT measures was associated with IQ (Gender and IQ distributions across classes are provided in the Tables A3 of the Appendix, the effects of gender and IQ on the questionnaire scores in Table A4 of the Appendix).

Table 3
Impact of gender and questionnaire scales on ConDAT measures [#]

Independent variable		*ConDAT variable*	*t test* [#]	*Bin. Log. Regr.; Exp(B)*
Female Gender		DTlog	1.812 (p=0.072)	(n.s. t-Test)
"		DTlog2	1.677 (p=0.095)	(n.s. t-Test)
"		DTlog3	1.937 (p=0.054)	(n.s. t-Test)
"		DTlog4	1.817 (p=0.071)	(n.s. t-Test)
CTRS	Anxious-Shy	PERSIST	1.855 (p=0.065)	(age adjusted score)
	Perfectionism	DTlog5	2.141 (p=0.034)	(age adjusted score)
	Hyperactivity	PERSIST	-1.765 (p =0.079)	(age adjusted score)
	DSM-IV: Hyperactivity	PERSIST	-2.257 (p=0.025)	(age adjusted score)
SDQ	Hyperactivity	PERSIST	-2.088 (p=0.038)	0.991 (p=0.039)
	"	DTlog5	-1.797 (p=0.074)	excluded
	Conduct Problems	DTlog	-2.221 (p=0.028)	excluded
	"	DTVAR	-2.111 (p=0.036)	excluded
	"	DTlog3	-2.255 (p=0.025)	excluded
	"	DTlog4	-2.363 (p=0.019)	excluded
	"	DTlog5	-3.068 (p=0.002)	0.476 (p = 0.031)
	"	(Age in Bin.Log.Regr.)	-	0.847 (p = 0.039)
	Total Difficulties	DTlog5	-2.374 (p=0.019)	excluded
	"	(Age in Bin.Log.Regr.)	-	0.807 (p = 0.002)
GR	Ambition	DTVAR	-2.322 (p=0.021)	0.991 (p = 0.023)
	Endurance	PERSIST	-1.985 (p=0.049)	excluded

Note. [#] Only significant results and trends reported; Positive/negative *t* values indicate positive/negative associations between ConDAT variables and independent variables; for abbreviations of the ConDAT variables see note in Table 2.

Effects of Psychopathology

The dichotomized questionnaire scores separated affected from non affected children. Differences in ConDAT scores between these categories were found in several variables (see Table 3). In particular hyperactivity was associated with less PERSISTENCE while anxiety was associated with more. DT in section 5 was positively associated with perfectionism, but negative with hyperactivity and conduct problems.

In the binary logistic regression analyses (Table 3) persistence but not DT in the two last trials remained a significant predictor of SDQ hyperactivity. Low age and low waiting time in the last section but no further ConDAT variables were predictive of SDQ Conduct Problems. High SDQ Total Difficulties category membership could be predicted by low age. Whereas high ambition could be predicted by a small DTVAR (see Table A3 in the Appendix for the distributions of the questionnaire scores and Table A4 in the Appendix for gender and IQ effects on the questionnaire scores).

3.5 Discussion

The current study investigated the willingness of children and adolescents to wait for a reward in an experiment in the face of diminishing returns per unit of time as the trial proceeded. The amount of the reward, which was delivered after the last trial, corresponded to the total of delivered token, and the delay of token delivery was identical to the rewarded time-on-task.

The ConDAT proved to be sensitive to age-related changes in delay behaviour in two ways. First, as predicted, age has a considerable effect on DT. The longer overall waiting times in older children confirmed the hypothesis of an increased tolerance to delay with age (Green, et al., 1994; Marco, et al., 2008; Walls, 1973). Second, PERSIST across trials which was not correlated to DT, was strongly dependent on age. Younger children were less tolerant of delay on average than older children and adolescents but also showed less persistence in their choices than the older participants.

There are several possible reasons for the stronger decrease of delay tolerance across the session in younger children: First, younger children may need more time to learn, how the task works and, therefore, under the assumption of a general inquisitiveness and motivation tend to underestimate the aversive component of the task at the beginning. Second, their less developed executive abilities, including time estimating, compared to older children also lead to an underestimation of the aversive component at the beginning of the task (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; McCormack, Brown, Maylor, Darby, & Green, 1999). Third, a more pronounced novelty effect in younger children, who are less experienced with computers than older children, may decrease their aversive feelings and therefore increase their delay tolerance as long as the novelty of the situation holds.

Given the independence of DT and PERSIST, we interpret these two measures as two independent components of delay tolerance: DT is a component independent from the passage of time, while PERSIST reflects the influence of the passage of time, which is larger the more the slope deviates from zero. This demonstrates the dynamic character of delay tolerance over time, with delay tolerance being less robust in younger children. Measures of DT variability within a session which are not only highly intercorrelated but also correlated with DT, do not provide important additional information, because they are neither associated with age, IQ or gender, nor with any of the psychopathology measures, except with the CTRS scale Conduct Problems. This is somewhat surprising given recent findings of increased variability in responding in ADHD (F. Xavier Castellanos, et al., 2005) perhaps suggesting that different mechanisms are controlling delayed responding than performance on tests of sustained attention or effort on cognitive tasks.

None of the main ConDAT variables (DT, PERSIST, DTVAR, DTFLUC) was associated with IQ in the present study. Studies on the association between IQ and delay report inconsistent findings. However, no positive association between IQ and an increased preference for immediate reward has been reported to our knowledge (Marco, et al., 2008; Olson, Hooper, Collins, & Luciana, 2007). Consequently, IQ may be negatively associated with preference for immediacy. The trend to longer waiting time in girls in our study is consistent with studies which reported either no gender effect (Olson, et

al., 2007; Wulfert, Block, Santa Ana, Rodriguez, & Colsman, 2002), or a rather small increased preference for immediate rewards in boys which has been interpreted in the context of evolutionary selection pressures in Silverman's meta analysis (2003).

Almost all differences in ConDAT scores found between the affected and non-affected groups according the questionnaire ratings of psychopathology are consistent with our hypotheses: Besides age, hyperactivity is the major cause for the diminishing DT across the session. We can interpret the strong decay of delay tolerance during the task as an indicator of a reduced ability of self-regulation characterizing both young normal children and children with ADHD. This analogy between younger age and ADHD suggest that ADHD is at least partly the expression of a delayed brain maturation, as supposed by structural brain imaging studies (F. X. Castellanos, et al., 2002; Shaw, et al., 2006).

Limitations

Despite the many advantages of the task, the results are affected by a large unexplained variance. One part of this variance is probably not task-specific but rather reflects the complexity and strong context sensitivity of motivational processes. For example, if the task was administered at the end of an exhausting EEG session found significantly shorter overall waiting times (data not published). Another important source of unexplained variance probably stems from positive reinforcing factors beyond the explicit reward. The most important evidence for implicit reinforcing factors is given by the fact, that the overall DT for the whole sample is sufficient for viewing 99.5% of the reward. Consequently, many of the children invested the largest part of their waiting time to an amount of the reward that lies below the perceivable limit; they aimed to maximise their performance instead of optimizing it. One part of this effect is the underside of the ability to measure delay tolerance across a wide range within a session. This was achieved by 'tuning down' the delivered token in pretests in order to provoke delay aversion even in subjects with a high delay tolerance. We suppose that intrinsic motivation representing personal goals like 'being the best', 'winning the game' etc. or social desirability account for much of this irrational uneconomic behaviour with respect to the reward.

Beside the characteristics of the task, several methodical constraints have to be considered. First our study was not designed to test for associations between delay aversion and psychopathology. The power to detect associations between psychopathology and delay choice is limited by the strongly skewed distribution with high frequencies at the unaffected end of almost all questionnaire scales. Another power limiting constraint results from small variation of questionnaire scores within classes, but large variation between classes, We assign a large part of this clustering within classes to differences in the teachers' perceptions or in their inner normative references. This cluster effect is further not reflecting different school types, because from grades one to six children are assigned to classes without considering their intelligence levels. Given all these limitations which reduce the power to detect associations between questionnaire measures and ConDAT variables, the findings probably are of higher validity than suggested by the statistics.

Conclusions

This study provides new insight into delay tolerance by decomposing it into a component which is independent from the passage of time (DT) and a component which is sensitive to the passage of time (PERSIST); components which are independent of each other but which both change with age. Furthermore these two delay related processes may have different developmental trajectories. The general willingness to wait for reward (independent component) particularly increases from late childhood to adolescence, while the ability to maintain an initial goal across the time-on-task (context sensitive component) predominantly develops in the earlier childhood. The associations between hyperactivity and delay choice support recent findings of delay aversion predicting ADHD (Müller, et al., 2009). Finally, the similarity of age effects and ADHD psychopathology effects on the change of the waiting times during the task provides some evidence for the theory of delayed brain maturation in children with ADHD.

3.6 References

Anderson, V. A., Anderson, P., Northam, E., Jacobs, R., & Catroppa, C. (2001). Development of executive functions through late childhood and adolescence in an Australian sample. Developmental Neuropsychology, 20(1), 385-406.

Antrop, I., Roeyer, H., Oost, P. V., & Buysse, A. (2000). Stimulation Seeking and Hyperactivity in Children with ADHD. Journal of Child Psychology and Psychiatry, 41(2), 225-231.

Asherson, P., & Image Consortium (2004). Attention-Deficit Hyperactivity Disorder in the post-genomic era. European Child & Adolescent Psychiatry, 13(1).

Barkley, R. A. (1998). Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment (2nd ed.): (1998). xii, 628 pp. New York, NY, US: Guilford Press.

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(8), 1793-1802.

Castellanos, F. X., Lee, P. P., Sharp, W., Jeffries, N. O., Greenstein, D. K., Clasen, L. S., et al. (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. Jama, 288(14), 1740-1748.

Castellanos, F. X., Sonuga-Barke, E. J. S., Scheres, A., Di Martino, A., Hyde, C., & Walters, J. R. (2005). Varieties of attention-deficit/hyperactivity disorder-related intra-individual variability. Biological Psychiatry, 57(11), 1416-1423.

Conners, C. K. (1997). Conners' Parent Rating Scale - Revised (L) / Conners' Teacher Rating Scale - Revised (L). New York: Multi-Health Systems Inc.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: a research note. Journal of Child Psychology & Psychiatry & Allied Disciplines, 38(5), 581-586.

Green, L., Fry, A. F., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. Psychological Science, 5(1), 33-36.

Heinrich, H., Moll, G. H., Dickhaus, H., Kolev, V., Yordanova, J., & Rothenberger, A. (2001). Time-on-task analysis using wavelet networks in an event-related potential study on attention-deficit hyperactivity disorder. Clinical Neurophysiology, 112(7), 1280-1287.

Kuntsi, J., Stevenson, J., Oosterlaan, J., & Sonuga-Barke, E. J. (2001). Test-retest reliability of a new delay aversion task and executive function measures. British Journal of Developmental Psychology, 19(3), 339-348.

Lin, C. C. H., Hsiao, C. K., & Chen, W. J. (1999). Development of Sustained Attention Assessed Using the Continuous Performance Test among Children 6–15 Years of Age. Journal of Abnormal Child Psychology, 27(5), 403-412.

Marco, R., Miranda, A., Schlotz, W., Melia, A., Mulligan, A., Mueller, U., et al. (2008). Delay and Reward Choice in ADHD: An Experimental Test of the Role of Delay Aversion. Neuropsychology, Manuscript in-press.

McCormack, T., Brown, G. D., Maylor, E. A., Darby, R. J., & Green, D. (1999). Developmental changes in time estimation: comparing childhood and old age. Developmental Psychology, 35(4), 1143-1155.

Mischel, W., & Ayduk, O. (2004). Willpower in a cognitive-affective processing system: The dynamics of delay of gratification. In R. F. Baumeister & K. D. Vohs (Eds.), Handbook of self-regulation: Research, theory, and applications (pp. 99-129). New York, NY: Guilford Press.

Mischel, W., & Baker, N. (1975). Cognitive appraisals and transformations in delay behavior. Journal of Personality and Social Psychology, 31(2), 254-261.

Mischel, W., Shoda, Y., & Peake, P. K. (1988). The nature of adolescent competencies predicted by preschool delay of gratification. Journal of Personality and Social Psychology, 54(4), 687-696.

Müller, U. C., Schönenberger, S., Sonuga-Barke, E., & Steinhausen, H. C. (2009). ADHD and Delay Sensitivity in Childhood and Adolescence: Waiting in the face of diminishing returns. Manuscript submitted for publication.

Müller, U. C., Sonuga-Barke, E. J., Brandeis, D., & Steinhausen, H.-C. (2006). Online measurement of motivational processes: introducing the Continuous Delay Aversion Test (ConDAT). Journal of Neuroscience Methods, 151(1), 45-51.

Olson, E. A., Hooper, C. J., Collins, P., & Luciana, M. (2007). Adolescents' performance on delay and probability discounting tasks: Contributions of age, intelligence, executive functioning, and self-reported externalizing behavior. Personality and Individual Differences, 43(7), 1886-1897.

Petry, N. M. (2002). Discounting of delayed rewards in substance abusers: relationship to antisocial personality disorder. Psychopharmacology, 162(4), 425-432.

Rapport, M. D., Tucker, S. B., DuPaul, G. J., Merlo, M., & Stoner, G. (1986). Hyperactivity and frustration: the influence of control over and size of rewards in delaying gratification. Journal of Abnormal Child Psychology, 14(2), 191-204.

Reynolds, B., Richards, J. B., Horn, K., & Karraker, K. (2004). Delay discounting and probability discounting as related to cigarette smoking status in adults. Behavioural Processes, 65(1), 35-42.

Sagvolden, T., Aase, H., Zeiner, P., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. Behavioural Brain Research, 94(1), 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. Behavioral & Brain Sciences, 28(3), 397-419; discussion 419-368.

Sattler, J. M. (1992). Assessment of Children: WISC III and WPSSI-R Supplement. Sandiego, CA: M. Sattler.

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(11), 2092-2103.

Seidel, W. T., & Joschko, M. (1990). Evidence of difficulties in sustained attention in children with ADDH. Journal of Abnormal Child Psychology, 18(2), 217-229.

Shaw, P., Lerch, J., Greenstein, D., Sharp, W., Clasen, L., Evans, A., et al. (2006). Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with attention-deficit/hyperactivity disorder. Archives of General Psychiatry, 63(5), 540-549.

Silverman, I. W. (2003). Gender Differences in Delay of Gratification: A Meta-Analysis. Sex Roles, 49(9-10), 451-463.

Sonuga-Barke, E. J. (2005). Causal models of attention-deficit/hyperactivity disorder: from common simple deficits to multiple developmental pathways. Biological Psychiatry, 57(11), 1231-1238.

Sonuga-Barke, E. J., Sergeant, J. A., Nigg, J., & Willcutt, E. (2008). Executive dysfunction and delay aversion in attention deficit hyperactivity disorder: nosologic and diagnostic implications. Child & Adolescent Psychiatric Clinics of North America, 17(2), 367-384.

Sonuga-Barke, E. J., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion--I. The effect of delay on choice. Journal of Child Psychology & Psychiatry & Allied Disciplines, 33(2), 387-398.

SPSS for Windows, Rel. 17.0.0, (2008). Chicago: SPSS Inc.

Tewes, U., Rossmann, P., & Schallberger, U. (2000). Hamburg-Wechsler-Intelligenztest für Kinder. Göttingen: Huber.

Walls, R. T. (1973). Delay of reinforcement development. Child Development, 44(3), 689-692.

Wulfert, E., Block, J. A., Santa Ana, E., Rodriguez, M. L., & Colsman, M. (2002). Delay of gratification: impulsive choices and problem behaviors in early and late adolescence. Journal of Personality, 70(4), 533-552.

4 Studie 3: ADHD and Delay Sensitivity in Childhood and Adolescence: Waiting in the face of diminishing returns.[1]

4.1 Abstract

Delay intolerance appears to be one of the core characteristics in attention-deficit / hyperactivity disorder (ADHD). Due to its large variation in sensitivity to delay in populations especially as a function of age and/or psychopathology, measuring delay intolerance is complicated by ceiling and floor effects – tasks designed for one group are to easy or too hard for other groups. The Continuous Delay Aversion Test (ConDAT) is a new computer task that utilizes a 'waiting under diminishing returns' scenario to overcome this limitation. This task was applied to a sample of 23 children with ADHD from age 7 – 15 and individually matched controls. Controls were willing to wait longer and showed less persistence in their choices over sessions than ADHD children. The good construct validity of the ConDAT was demonstrated by 72% correctly classified participants. The lower delay tolerance and its persistence over session in ADHD children provides evidence for delay aversion in ADHD.

[1] Müller, U. C., Schönenberger, S., Sonuga-Barke, E., & Steinhausen, H. C. (2009). ADHD and Delay Sensitivity in Childhood and Adolescence: Waiting in the face of diminishing returns. Manuscript submitted for publication.

4.2 Introduction

On a theoretical level, it has been suggested that Attention-deficit/hyperactivity disorder ADHD symptoms are caused mainly by deficits in executive functioning (Barkley, 2004), which can be defined as higher order processes regulating and controlling lower order processes and are involved in goal- and future related behaviour (Alvarez & Emory, 2006; Nigg, Goldsmith, & Sachek, 2004). Consistent with this assumption, deficits in response inhibition, working memory, planning, and set shifting have been identified in ADHD (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). However, executive deficits may be specific to inattentive symptoms symptoms (Willcutt, et al., 2005), and a substantial proportion of ADHD / Combined Tye patients have no executive deficits (Doyle, et al., 2005; Nigg, et al., 2005).

Alternative models of ADHD include the deficient response to reward resulting in a decreased effectiveness of reinforcers (Luman, Oosterlaan, & Sergeant, 2005; Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). The latter has been conceptualized, e.g., as a need for abnormal high reinforcement rates (Haenlein and Caul (1987), a shortened and steeper delay-of-reinforcement gradient (Sagvolden & Sergeant, 1998), an unusually low frustration threshold (Douglas & Parry, 1994), or a greater intolerance for delay (Sonuga-Barke, Taylor, Sembi, & Smith, 1992). Recent approaches attempt to integrate both cognitive and motivational aspects in one framework of ADHD e.g. by contrasting executive inhibition with motivational inhibition (Nigg, 2001), or hot with cold executive functions (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006). Sergeant's cognitive energetic model (2000; 2005) emphasizes a hierarchical structure between the level of primary attention functions, the level of energetic resources, and the superordinated executive level.

The dual pathway theory (Sonuga-Barke, (2002, 2005) regards ADHD as a neuro-psychologically heterogeneous disorder with a cognitive pathway mediated by inhibitory control deficits, and a motivational pathway mediated by delay aversion, both regulated mainly by dopamine transmission. The cognitive pathway postulates a dysfunction of executive regulatory brain circuits assuming a basal inhibition deficit leading to executive dysfunction which in turn causes ADHD symptoms. In contrast,

the motivational pathway implicates dysfunctions of reward circuits leading to an abnormal weak representation of temporal distal reinforcers. This abnormality causes motivational deficits in delay tolerance from which secondarily symptoms of inattention, hyperactivity or impulsivity can arise.The more elaborate version of the dual pathway model additionally integrates environmental and learning aspects: punishing responses to impulsive behaviour can establish and stabilize a general delay aversion, which secondarily exacerbates inattentive, hyperactive, or impulsive behaviour. Furthermore, the failure to engage in delay rich situations restricts the development of adaptive skills that normally help to manage delay (Sonuga-Barke, 2005). An intolerance for delay in children with ADHD has been shown in many studies (Marco, et al., 2008; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986; Sagvolden, Aase, Zeiner, & Berger, 1998; Sonuga-Barke, et al., 1992), but not in all studies (e.g. Scheres, et al., 2006) probably due to differences in the applied methods (Scheres, et al., 2006).

The negative impact of delay on the response rewards is, in fact, not specific to ADHD but a form of the common phenomenon of temporal discounting (Green, Fry, & Myerson, 1994). Because the value of future rewards is discounted as they are delayed it is often difficult, even for people without ADHD or other forms of psychopathology, to choice in favour of important long-term goals in mind when faced with short-term gratifications (Mischel & Ayduk, 2004). The abilty to delay gratification in this way is known to develop with age (Green, Myerson, Lichtman, Rosen, & Fry, 1996), to be probably more pronounced in females (Silverman, 2003), and to be reduced as a function of substance abuse (Petry, 2002; Reynolds, Richards, Horn, & Karraker, 2004).

The measurement of delay tolerance is complicated by the very rapid changes that occur in both delay tolerance and reward salience as children grow older (Green, et al., 1994; Olson, Hooper, Collins, & Luciana, 2007; Walls, 1973), and by variation that is exacerbated by the above described psychopathology. This makes it unlikely that any individual task can validly assess delay sensitivity over widely different ages and across various psychopathological conditions. Tasks designed for young children will probably show ceiling effects when used with older children. For instance, it has been suggested that a task commonly used for indexing delay sensitivity in childhood, the Maudsley Index of Delay

Aversion (MIDA; Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001b), which has been used in different studies before (Antrop, et al., 2006; Kuntsi, Oosterlaan, & Stevenson, 2001a; Sonuga-Barke, et al., 1992), is inappropriate for adolescents. This is due to the limited range of sensitivity of the test so that it produces large ceiling effects (Marco, et al., 2008) which are caused to a large extent by the well-known general increasing tolerance to delay with age (Green, et al., 1994).

To overcome these and other constraints in measuring delay tolerance, Müller and colleagues (2006) developed the Continuous Delay Aversion Test (ConDAT), which is used in the present study. The ConDAT was designed specifically to have sensitivity across a wide range rewarded of delay tolerances, allowing it to quanity individual sensitivity yo reward from the most intolerant (those who dislike even the shortest delay and those who are prepared to tolerate even the longest delay for minima measure changes in delay preferences across trials, and uses adaptive rewards of an emotional but not material character. Additionally, according to postulated criteria for neuroscientific research in ADHD (Castellanos & Tannock, 2002), it provides a dimensional output, can be used in an EEG-settings, and has a satisfactory test-retest reliability (Müller, et al., 2006).

Like other delay choice tasks, the ConDAT positively associates the amount of the reward with its delay. However, instead of offering the choice between two fixed choice options at the beginning of each trial, the task continuously delivers a stream of token rewards and participants are asked to say at which point they want to stop the trial and get the accumulated reward. It is crucial to the test that each trial incorporates a diminishing returns scenario by reducing the delivery rate of rewards as the trial progresses. The rate is far higher in the beginning than at the end of each trial. Thus, this reduction in value for each time unit should heighten the sensitivity to delay especially towards the end of the trial so that even the most delay tolerant individual would be challenged to show some delay aversion. A more detailed description will be provided in the methods section and in the appendices. Previous studies have demonstrated the test-retest reliablility (Müller, et al., 2006) and broad sensitivity (Müller, Sonuga-Barke, & Steinhausen, 2009) of the task. The well-known increasing tolerance to delay with increasing age should be reflected in longer mean waiting times in older than in younger

children. In addition, with respect to the developing executive skills, particularly, planning and time estimation older children should show more stable, i.e., less decreasing waiting times across the session than younger children. Both effects were demonstrated in another study using the same task in a large normal sample (Müller, et al., 2009).

The current study had four main aims. First to explore the limits of delay tolerance in ADHD children using the ConDAT. The predictive validity of the ConDAT task was also examined. Second, to test the hypothesis that children with ADHD suffer a developmental lag with regard to delay-related behaviour. Are ADHD children comparable to younger control children with respect to delay tolerance. If this would be true, both ADHD and younger age should reduce the overall tolerance to delay and both should be associated with decreasing waiting times with time-on-task. Third, to examine the extent to which patterns of responding to delay established at the start of the trial persist across the trial. ADHD children have in the past been shown to show a difficulty sustaining effort over time of demanding tasks (Heinrich, et al., 2001; Seidel & Joschko, 1990). Is this effect likely to be seen with regard to delay tolerance. Finally, the effects of IQ, gender, and comorbidity on the ConDAT performance of ADHD children were explored.

4.3 Methods

4.3.1 Participants and recruitment

Twenty-three children (M = 10.6 years; 3 girls) with ADHD and 23 normal control children matched by age and gender participated in the study. The ADHD group was recruited from the specialist ADHD Centre of the Department of Child and Adolescent Psychiatry, University of Zürich. Children were included into the case group if they had a clinical diagnosis of ADHD, were within the age range of 6 – 16 years, and had an IQ of at least 80. Additionally, in both the inattention and hyper

activity/impulsivity domain, six or more symptoms as measured by the Conners Parents and Teacher Rating Scales (CPRS-R:L, CTRS-R:L) had to be present (Conners, 1997a, 1997b). Alternatively, theT-score in the corresponding Conners DSM-IV scales (DSM-Inattention and DSM-Hyperactivity/Impulsivity) had to be at least 65. Three of the 35 candidate children for the ADHD group were excluded because they did not fulfil clinical diagnostic criteria, and nine because they did not meet the cut-off criteria of the questionnaires. Seven of the remaining 23 patients were treated with methylphenidate.

The control children were identified from an existing community sample of 183 children (M = 11.3 years; age range 6.1 – 16.9; 93 girls) which was recruited from 9 school classes. In the control sample, only teacher versions of the questionnaires were available. Inclusion into the control group was restricted to children with no more than two symptoms on the CTRS inattention and hyperactivity/impulsivity domains and T-scores of less than 60 on the CTRS-R:L scales covering ADHD symptoms, i.e., the scales B, C, and H to N. Additionally, both the Total Difficulties Score and the Hyperactivity Score of the Strength and Difficulty Questionnaire (SDQ, Goodman, 1997) had to above the "affected" cutoff (i.e., 16 and 7 for the scales Total Difficulties and Hyperactivity, respectively; see www.sdqinfo.com). 52 boys and 54 girls fulfilled these criteria and were available as potential controls for this study.

Individual matching of the 23 ADHD children and the 23 controls was based on the following hierarchical rules: 1) identical gender 2) smallest individual age difference 3) smallest mean age difference between groups, and 4) random selection. It was not necessary to make use of the random criteria. The final samples were equal in size and gender (each n = 23; 3 girls). The age difference within pairs was at most 4 months, except for two pairs (7 months), and both samples had a mean age of 10.6 years (see Table 1).

Table 1
Intelligence and questionnaire findings in the two groups

	ADHD				Controls				ADHD – Controls / ES			
Intelligence	*Mean*	*SD*	*Min*	*Max*	*Mean*	*SD*	*Min*	*Max*	*Mean*	*ES*	t^{paired}	
IQ #	102.5	8.6	89	121	109.7	12.4	87	136	-7.2	-0.58	-1.89	n.s.
Vocabulary #	10.6	2.6	6	17	11.4	2.0	8	15	-0.8	-0.39	-1.00	n.s.
Similarities	11.3	2.3	8	16	12.0	2.2	8	15	-0.7	-0.34	-1.23	n.s.
Block Design #	9.9	2.0	7	16	12.6	3.4	3	17	-2.7	-0.79	-2.89	**
Picture Completion	9.7	2.3	5	14	10.1	3.1	5	17	-0.4	-0.12	-0.47	n.s.
Digit Span #	9.2	2.2	6	13	9.7	2.8	4	15	-0.5	-0.17	-0.76	n.s.

	Teacher ratings											*Parent ratings (ADHD)*				*Teacher - Parents (ADHD) / ES*			
Conners rating scales	*Mean*	*SD*	*Min*	*Max*	*Mean*	*SD*	*Min*	*Max*	*Mean*	ES^{contr}	$Z^{Wilcoxon}$	*Mean*	*SD*	*Min*	*Max*	*Mean*	ES^{pooled}	$Z^{Wilcoxon}$	
A. Oppositional	68.1	11.6	51	89	47.0	4.3	45	65	21.1	4.96	-4.03 ***	65.7	9.2	47	82	2.4	0.29	-1.20	n.s.
B. Cognitive Problems	63.3	9.8	44	77	46.0	4.1	41	54	17.3	4.19	-4.11 ***	67.4	9.6	49	88	-4.1	-0.58	-1.88	n.s.
C. Hyperactivity	69.8	10.1	48	90	46.0	2.9	43	53	23.7	8.23	-4.20 ***	68.4	11.1	49	87	1.3	0.15	-0.59	n.s.
D. Axious-Shy	62.7	13.2	42	87	54.2	7.5	45	75	8.5	1.14	-2.34 *	57.6	13.3	41	89	5.1	0.71	-1.45	n.s.
E. Perfectionism #	63.7	11.1	49	87	59.4	9.8	44	79	4.3	0.44	-1.18	n.s.	62.7	10.9	44	90	1.0	0.18	-0.90	n.s.
F. Social Problems °	60.3	14.7	45	88	49.8	10.0	45	88	10.5	1.05	-3.23 **	62.7	13.6	45	90	-2.4	-0.30	-0.61	n.s.
G. Psychosomatic												61.3	15.4	42	90				
H. ADHD Index	70.7	8.8	49	89	44.1	3.2	41	53	26.6	8.43	-4.20 ***	68.5	8.7	53	83	2.2	0.25	-0.75	n.s.
I. GI: Restless-Impulsive	69.8	8.7	52	90	44.5	3.9	41	55	25.3	6.50	-4.20 ***	70.3	10.7	52	90	-0.5	-0.06	-0.05	n.s.
J. GI: Emotinal Lability	64.8	14.2	45	90	46.1	3.3	44	57	18.7	5.66	-4.11 ***	58.7	12.6	42	85	6.1	0.61	-1.51	n.s.
K. GI: Total	70.0	9.6	53	90	44.4	3.4	41	54	25.7	7.66	-4.20 ***	68.2	11.2	50	87	1.9	0.22	-0.67	n.s.
L. DSM: Inattention	66.9	9.3	51	86	44.7	3.2	40	51	22.2	7.04	-4.20 ***	68.0	10.0	46	89	-1.1	-0.14	-0.49	n.s.
M. DSM: Hyperactive/Impulsive	69.7	10.6	47	88	45.4	3.4	42	56	24.4	7.12	-4.20 ***	67.5	10.0	50	86	2.3	0.26	-0.54	n.s.
N. DSM: Total	70.1	8.2	53	89	44.8	2.7	41	51	25.3	9.39	-4.20 ***	69.4	9.5	48	85	0.7	0.08	-0.26	n.s.
SDQ	*Mean*	*SD*	*Min*	*Max*	*Mean*	*SD*	*Min*	*Max*	*Mean*	ES^{contr}	$Z^{Wilcoxon}$	*Mean*	*SD*	*Min*	*Max*	*Mean*	ES^{pooled}	$Z^{Wilcoxon}$	
Total Problem score	18.4	5.8	8	30	4.7	4.0	0	13	13.7	3.43	-4.17 ***	17.1	6.5	6	28	1.3	0.28	-0.93	n.s.
Emotional Symptoms °	3.0	2.4	0	9	0.8	1.5	0	6	2.3	1.50	-3.00 **	3.2	2.6	0	10	-0.1	-0.07	-0.20	n.s.
Conduct Problems	4.2	2.1	0	9	0.7	1.0	0	3	3.5	3.45	-4.14 ***	4.0	2.4	1	9	0.3	0.09	-0.51	n.s.
Hyperactivity	7.6	1.9	2	10	1.6	1.4	0	4	6.0	4.29	-4.12 ***	6.8	2.1	3	10	0.8	0.25	-1.68	n.s.
Peer Problems	3.5	2.6	0	9	1.4	2.2	0	7	2.1	0.96	-2.34 *	3.2	2.4	0	8	0.3	0.17	-0.26	n.s.
Prosocial Behaviour	3.4	2.1	0	7	6.7	2.5	3	10	-3.4	-1.36	-3.67 ***	7.3	1.5	5	10	-3.9	-10.88	-4.03	***

Note: # N of ADHD group = 22 ° N of Control group = 22 ES^{contr} Effect size with variance of controls ES^{pooled} Effect size with Pooled Variance t^{paired} Paired *t* test $Z^{Wilcoxon}$ *Wilcoxon-Test (Z)*
* p <0.05 ** p<0.01 *** p<0.001

4.3.2 Measures

Delay Tolerance Task

The basic idea of the ConDAT has already been described in the introduction. The task is programmed in the code of the Presentation® Software (see http://nbs.neuro-bs.com) and runs on a conventional computer with Windows® OS 98 and higher. The computer program includes all parts of the task, i.e. instruction, three practising trials, 30 main trials, and the rewarding video clip.

The program provides a very detailed logfile, which includes a precise temporal listing of all stimuli and response occurrences. The most important primary output parameter is the rewarded waiting time in each trial representing the amount of delay a person is willing to accept with regard to the corresponding reward.

The reward size is based on all periods of the session which actually are accessible for collecting the token from the subject's view. Consequently, the 30 feedback trials and the decision periods, i.e., the time a subject needs to consider and perform a response to the programmed repeated freezing of the rewarding process (see Figure 1) are not rewarded. The average delay time was a measure of delay tolerance (DT) while the slope of the change in DT was also calculated and represented a measure of persistence of DT (PERSIST). Both DT and PERSIST are reported in seconds per trial.

A third measure (CLICKS) counts the number of mouse clicks which are applied beside the functional purpose of releasing the token delivery after the programmed freezings (see Figure 1 and Appendix 1). These useless mouse responses without any effect on the task are calculated by subtracting the functional mouse responses from the totally recorded mouse responses. (see Appendices for a detailed description of the task and sample data).

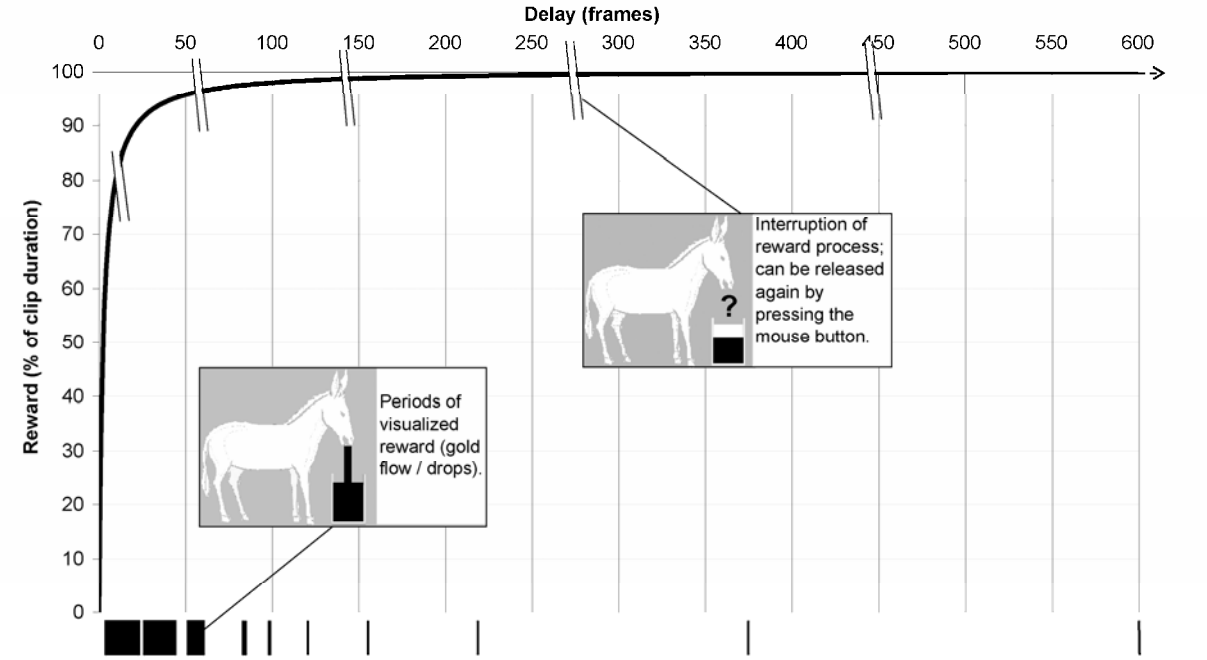

Figure 1 Reward mechanism of the ConDAT. The curve indicates the cumulative amount of returned token during a trial (1 frame = 1/10 sec.). This is visualized by a steadily diminishing and more and more interrupted stream of gold out of a donkeys mouth into a container. The black bars at the bottom of the figure indicate the phases of actual gold flow which is adapted to the slope of the curve. The token delivery is interrupted from time to time (double backslashes) and can be reactivated by pressing a mouse button. Each trial can be terminated at any time by pressing the spacebar.

Questionnaires

All questionnaires were completed by the teachers of all children and by the parents of the probands. The 59 items of the Conners' Teacher Rating Scale (CTRS-R:L) are summarized into the following 14 scales: (A) Oppositional, (B) Cognitive Problems/Inattention, (C) Hyperactivity, (D) Anxious/shy, (E) Perfectionism, (F) Social Problems, (H) Conners' ADHD Index, (I) Conners' Global Index: Emotional, (J) Conners' Global Index: Impulsivity, (K) Conners' Global Index: Total, (L) DSM–IV ADHD Symptoms: Inattention, (M) DSM–IV ADHD Symptoms: Hyperactivity/Impulsivity, and (N) DSM–IV ADHD Symptoms. The Conners' Parents Rating Scale has 80 items and an additional scale, (G) Perfectionism. The raw scores of both Conners' questionnaires are transformed into T-scores using the US standardization sample.

Both teacher and parents' versions of the SDQ summarize 25 questions into the five following scales: Emotional Symptoms Score, Conduct Problems Score, Hyperactivity Score, Peer Problems Score, Prosocial Behaviour Score, and the Total Problems score summarizing all scores except the Prosocial Behaviour score.

Intelligence

The five used subtests of the German version of the WISC-III (Tewes, Rossmann, & Schallberger, 2000) are Vocabulary, Similarities, Block Design, Picture Completion, and Digit Span. The IQ was prorated from these subtests except Digit Span, following a procedure introduced by Sattler (1992).

4.3.3 Data transformation and analyses

The variables DT and PERSIST and all IQ measures were normally distributed (Kolmogorov-Smirnov-Z = n.s.). The distribution of the variable CLICK was clearly skewed and bimodal and therefore it was dichotomized (CLICKCAT) using a cutoff score of 50 so that there were high responders (N = 5, mean = 115.4(21.5), range = 82 – 141) and low responders (N = 41, mean = 8.2(9.0), range = 0 – 42). Almost all questionnaire measures were markedly skewed and, therefore, only non-parametric analyses were performed. In order to investigate possible nonlinear effects of age on ConDAT measures, the sample was divided into a younger group (11 boys and 1 girl) and an older group (9 boys and 2 girls). No matched pair was separated by this procedure.

In a first step the effect of IQ and gender on three ConDAT measures DT, PERSIST and CLICKCAT was analysed by performing a Two-Way-MANOVA with IQ and gender as independent variables. In a second step Three-Way ANCOVAs were performed to assess effects of age group and condition on the three main ConDAT measures DT, PERSIST and CLICKCAT. IQ, which had a significant effect

of DT in the first step analysis, was entered as a covariate. Binary logistic regression (foreward conditional method) with condition as dependent variable and all ConDAT measures as predictors was employed to estimate the predictive power of the ConDAT. The effect of comorbid conditions on ConDAT measures was analyzed by use of nonparametric correlations and, in a second step, three ANCOVA's with each DT, PERSIST and CLICKCAT as dependent variables and the significantly correlated comorbidity measures plus condition as covariates are performed. All statistics were calculated by the use of the SPSS program (2008).

4.3.4 Procedure

All children of the ADHD group and their parents gave written informed consent prior to the study. Parents of the control children were informed by the teachers and also gave written consent. All participating children orally agreed to participate in the study. Whereas all children of the ADHD group were assessed in the clinic, control group children were assessed at school in a separate room next to the class room.

The last dose of methylphenidate was administered at least 24 hours before the assessment session, which included the delay choice task and the short form of an IQ test. Because the ConDAT is highly sensitive to motivation and fatigue, it was always administered at the beginning of the session. The main part of the ConDAT was preceded by a step by step introduction including the selection of the reward, and three practising trials; the playback of the rewarding video clip immediately followed after the last trial had finished. The experimenter had to make sure that the proband was familiar with the principle and the functioning of the task, before starting the 30 main trials. A more detailed description of the task procedure is provided in Appendix II.

4.4 Results

The MANOVA of IQ and gender on the three ConDAT measures DT, PERSIST and CLICKCAT revealed no multivariate effects of IQ or gender. However, there was a significant univariate effect in the corrected model on DT ($F = 3.505$, $p = 0.039$) due to a significant effect of IQ on DT ($F = 5.799$, $p = 0.020$). Consequently, IQ was entered as a covariate in the subsequent ANCOVA of age group and condition on DT.

When DT was the dependent variable, a lower delay tolerance of children with ADHD was confirmed by a significant main effect of condition on DT. No other main or interaction effect on DT was significant. The lower DT score in ADHD children was large effect size of 0.8 (Table 2).

When PERSIST was the dependent variable, there was a main effect of age group ($F = 4.786$, $p = 0.035$) and an interaction effect of age group x condition ($F = 6.422$, $p = 0.015$). Older children scored 0.362 points higher on PERSIST, indicating that their waiting time increased 0.3 seconds more per trial when compared to the younger children. This difference was pronounced and significant in control children, where older children increased 0.074 seconds more per trial compared to the ADHD children, corresponding to an increase in waiting of 14.9 seconds from the first to the last trial in older children, but a decrease of 7.4 seconds in younger children (see Figure 2). Both ADHD groups, in contrast, had similar, stable and not significantly differing waiting times across the task, corresponding to a very small increase of 0.2 seconds across the task in older children and an increase of 0.7 seconds in younger children, as shown in Table 2 and Figures 2 and 3.

When CLICKCAT was the dependent variable, there was a main effect of condition ($F = 6.066$, $p = 0.018$), but not of age group, and no interaction effect. All children from the category of extreme responders belonged to the ADHD group.

Table 2
ConDAT measures findings

Total sample	***All***				***ADHD***				***Controls***				***Differences***		
	Mean	*SD*	*Min*	*Max*	*Mean*	*SD*	*Min*	*Max*	*Mean*	*SD*	*Min*	*Max*	*Mean*	*E.S.*	*Statistic*
DT #	22.12	12.53	5.52	59.17	16.63	8.48	7.24	32.48	27.62	13.65	5.52	59.17	-10.99	-0.8	*t* -3.052 **
PERSIST #	0.062	0.572	-1.288	2.259	0.015	0.306	-0.692	0.771	0.110	0.756	-1.287	2.259	-0.095	-0.1	*t* -0.509
CLICK	19.87	35.36	0	141	30.8	47.1	0	141	9.0	9.7	1	42	21.8	2.3	*Z* -1.724
CLICKCAT (high)	5/46				5/23				0/23				5		χ^2 5.490 *
Younger subgroup															
DT #	20.91	11.43	7.24	43.02	16.73	8.88	7.24	30.23	25.08	12.50	9.42	43.02	-8.36	-0.7	*t* -1.755
PERSIST #	-0.111	0.509	-1.288	0.902	0.024	0.356	-0.692	0.771	-0.246	0.612	-1.288	0.902	0.27	0.4	*t* 1.115
CLICK	28.33	44.75	1	141	46.8	57.5	1	141	9.8	11.4	1	42	37.0	3.2	*Z* -2.121 *
CLICKCAT (high)	4/24				4/12				0/12				4		χ^2 4.800
Older subgroup															
DT #	23.45	13.78	5.52	59.17	16.52	8.44	7.66	32.48	30.38	14.90	5.52	59.17	-13.86	-0.9	*t* -2.495 *
PERSIST #	0.251	0.589	-0.458	2.260	0.005	0.257	-0.458	0.526	0.498	0.727	-0.124	2.26	-0.493	-0.7	*t* -1.952
CLICK	10.64	17.76	0	82	13.3	24.2	0	82	8.0	7.8	1	25	5.3	0.7	*Z* -0.359
CLICKCAT (high)	1/22				1/11				0/11				1		χ^2 1.048
Difference (older - younger)															
DT #	2.547	2.354	-1.714	16.15	-0.20	-0.44	0.42	2.26	5.30	2.40	-3.89	16.15	-5.50		
PERSIST # (*ti*)	0.362	0.080	0.830	1.358	-0.019	-0.098	0.234	-0.246	0.744	0.115 *	1.164	1.358	-0.762		
CLICK	-17.69	-26.98	-1	-59	-33.6	-33.3	-1	-59	-1.8	-3.7	0	-17	-31.7		
CLICKCAT (high)	-3				-3				0						

Note: DT = Average time over trials invested in collecting token PERSIST = slope of the regression line of DT on trials CLICK = number on unnecessary mouse responses CLICKCAT = dichotomized CLICK variable (cutoff value = 50) # in seconds E.S. = effect size with variance of controls *t:* paired *t* test, *Z:* Wilcoxon-Test, χ^2*:* Chisquare Test *ti* : independent *t* test * p <0.05 ** p<0.01

In a binary logistic regression analysis with DT, PERSIST, and CLICKCAT as predictors and condition as outcome, DT was entered in a first step into the equation (Wald statistics = 7.663, p = 0.006), and CLICKCAT (Wald statistics = n.s.) was added to DT (Wald statistics = 6.936, p = 0.008) in a second step. Removing the constant term from the model led to significant changes of the -2-log-likelihood in the second step for both DT (9.65, p = 0.002) and CLICKCAT (7.69, p = 0.006).

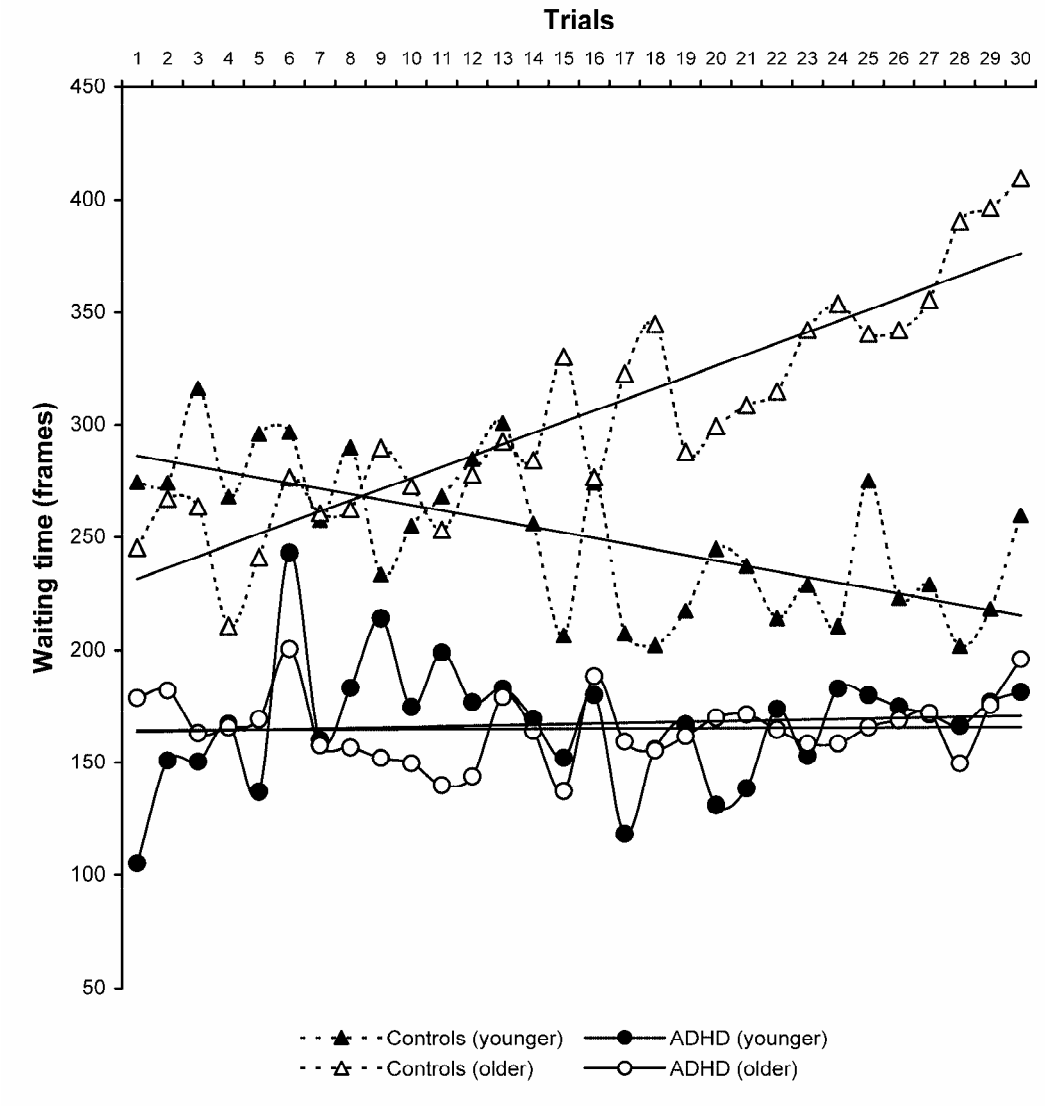

Figure 2 *Effects of condition and age group on reward collecting over a session of 30 trials of the ConDAT. The net waiting time in frames of 1/10 seconds corresponds to the overall trial time minus all non-rewarded segments which are the feedback trials and the periods between the repeated stopping of the token and its release by the proband.*

DT alone (first step) resulted in sensitivity of 65 percent and specificity of 61 percent and was able to classify correctly 63 percent of the participants. By adding CLICKCAT in the second step sensitivity increased to 74 percent and specificity to 70 percent resulting in 72 percent of correctly classified participants.

Table 3 shows the nonparametric correlations between various comorbidity scales and the ConDAT scores. The pattern of correlations was clearly different in children with ADHD and controls. In the combined groups, DT was the ConDAT parameter that was most sensitive to comorbid conditions which were all associated with lower delay tolerance.

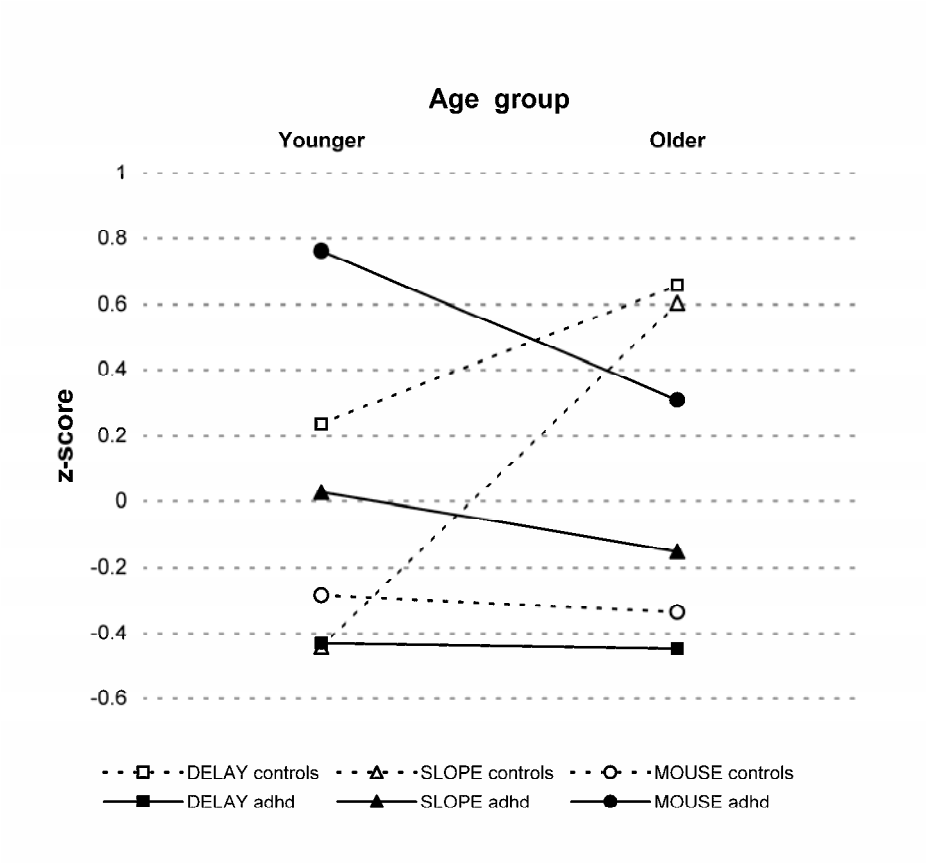

Figure 3 *Standarized scores of average waiting time (DELAY), change of waiting time (SLOPE), and unnecessary mouse responses (MOUSE) across age groups and ADHD conditions.*

Table 3

Correlations comorbid symptoms with ConDAT measures [#]

	ConDAT measures								
	DT			PERSIST			CLICK		
	Controls	*ADHD*	*All*	*Controls*	*ADHD*	*All*	*Controls*	*ADHD*	*All*
CTRS Oppositional	-	-	-.267 °	-.501 *	-	-.269 °	-	-	-
CTRS Anxious-shy	-	-	-.283 °	-	-	-	-.454 *	-	-
CTRS Perfectionism	-.557 **	-	-.482 **	-	-	-	-.402 °	-	-
CTRS Social Problems	-	-	-	-	-	-	-	-	-
CTRS Emotional Problems	-	-.293 *	-	-	-	-	-	-	-
SDQ(T) Emotional Symptoms	-	-	-.317 *	-	-	-	-.361 °	-	-
SDQ(T) Conduct Problems	-	.489 *	-	-.403 °	-	-	-	-	-
SDQ(T) Peer Problems	-	-	-.297 *	-	-	-	-	-	-
SDQ(T) Prosocial Behaviour	-	-	.267 °	-	-	-	-	-.427 *	-.281 °

Note: [#] Spearman's reank correlations; only trends and significant correlations are reported CTRS = Conners Teacher Rating Scales SDQ(T) = Teacher version of the Strenghts and Difficulties Questionnaire ° p <0.1 * p <0.05 ** p<0.01 *** p<0.001

In the subsequent ANCOVA's with DT as dependent variables and all significant correlating questionnaire scores plus condition as predictors, only the effects of perfectionism ($F = 10.962$, $p = 0.002$) and of condition ($F = 4.704$, $p = 0.037$) remained significant, corresponding to lower mean waiting times in children with high perfectionism and in the ADHD group. In both ANCOVA's with PERSIST and CLICK as dependent variable no effect remained significant.

4.5 Discussion

Children with ADHD were less willing to tolerate delay than controls when confronted with a diminishing reward return over each trial of the ConDAT task. This effect is comparable to findings in other studies on delay sensitivity (Marco, et al., 2008; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986; Thorell, 2007; Tripp & Alsop, 2001). The effect size of 0.8 is higher than effect sizes found in most of the measures covering not only motivational, but also executive deficits in ADHD (Kuntsi, Oosterlaan, & Stevenson, 2001; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). The ability to allocate effort for maintaining long term goals in mind and resisting to immediate rewarding behaviour is crucial in almost any situation of explicit learning not only of academic matters but also of skills and abilities (Mischel & Ayduk, 2004). Therefore, it was assumed that a large part of school problems in ADHD children arise from difficulties in tolerating delay (Solanto.2001) so that the present findings are of ecological relevance.

Ceiling and floor effects were absent in the almost nomally distributed DELAY variable indicating that the range of sensitivity of the task was not challenged by the probands. Based on findings in another study with the same task (Müller, Sonuga-Barke, & Steinhausen, 2009) and further studies as well (e.g. Green, Fry, & Myerson, 1994; Marco, et al., 2008; Olson, Hooper, Collins, & Luciana, 2007; Scheres, et al., 2006) we expected age to be a major source of variation in our sample. However, probably due to the large variation between the ADHD groups and the control group and the relative small sample size we found no main effects of age on the mean waiting time. Furthermore, the reward

concept of offering a video clip of choice probably minimized the variance in reward salience and value which is normally present in delay choice tasks (Green, Myerson, Lichtman, Rosen, & Fry, 1996; Marco, et al., 2008; Rapport, et al., 1986).

The amount of change in the waiting times across trials, as measured by the SLOPE variable, did not differ between controls and children with ADHD. Ths finding was in contrast to our expectation of a higher decrement of the willingness to wait for reward during the task in children with ADHD. These expectations were based on several considerations. First, novel situations are known to decrease hyperactive behaviour, a phenomenon which has been explained by an increased stimulation of the attention system (Zentall & Zentall, 1983) or a lack of responses to be reinforced in a new situation (Sagvolden, Wultz, Moser, Moser, & Morkrid, 1989). The situation at the beginning of the task should also reduce the subjective experience of delay, because a higher level of stimulation decreases the awareness of time (Sonuga-Barke, 1994). Second, there is evidence for contextual factors influencing delay tolerance (Rapport, et al., 1986; Tripp & Alsop, 2001). Further, the experienced delay may cumulatively increase aversive feelings with passage of time which is analogue to the decreasing sustained attention with time-on-task in children with ADHD (Heinrich, et al., 2001; Seidel & Joschko, 1990). Finally, Müller et al. (2009) found a lowered SLOPE in children of a community sample who scored high in hyperactivity measures.

Furthermore, against our expectations we found no age effects on SLOPE, particularly, no evidence for better executive skills in older children, which may have led to more stable waiting times in this age group and no evidence for a larger novelty effect in younger children, who are less familiar with computers and, therefore, may become less delay averse at the beginning of the task (Carlson, Mann, & Alexander, 2000). However, these expected effects were not completely missing. As the interaction effect between age group and condition on SLOPE suggests, these effects were absent only in children with ADHD who independently of age were insensitive to the temporal context.

n contrast, the controls showed these effects of a decreasing delay tolerance with time-on-task in younger age. However, with increasing age they maintained or even increased tolerance. This age effect in controls may be interpreted generally as reflecting the maturation of executive functions (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; Barkley, 1997; McCormack, Brown, Maylor, Darby, & Green, 1999). In contrast to younger children with less developed executive capacities, older children are more skilled to evaluate temporal aspects of the task and its relation to the own capacity and, thus, avoide an underestimation of the task requirements with respect to the own resources.

The fact that DELAY and SLOPE were not correlated provides evidence for two rather independent components of delay aversion: DELAY as a component reflecting a general delay tolerance irrespective of temporal context, i.e. of time-on-task, and SLOPE as a component that is sensitive to time-on-task. The question whether one or both of these components may help in classifying children according to the three categories which have been suggested by Sonuga-Barke (1992), namely, impulsive, reward maximizing, or delay averse, cannot be answered properly because we used only the version with fixed trial numbers and no post-reward delay.

However, the theoretical framework of the dual pathway model (Sonuga-Barke, 2005) may help to interpret our findings. Delay aversion in normal children may occur in delay rich situations depending on a general delay tolerance that increases with age. Additionally, probably due to a smaller repertoire of compensating self regulatory strategies in younger age, e.g. self distracting ‘cooling strategies’ (Mischel & Ayduk, 2004), younger children seem to be more vulnerable to contextual factors like time-on-task resulting in a diminishing delay tolerance with with task duration. The lack of age effects on both DELAY and SLOPE in ADHD children resulting in consistently low waiting times across the task and independent on age provides evidence for a non-adaptive chronic delay aversion which may include an individual learning history of negative environmental reactions to failures under delay condition and a secondary deficit in compensatory strategies due to reduced experience of coping with delay (Sonuga-Barke, 2005).

This more comprehensive model of behaviour shown by children with ADHD in delay-rich situations confirms the complexity of cognitive-affective processes underlying choices for or against delay. Mischel and Ayduk (2004) described these self-regulating processes as subjective calculations that include encoding processes, e.g., the subjective meaning and the self-relevance of the situation, activation of cognitive-affective representations like expectations and believes, affective reactions, individual values and goals etc. but also self-efficacy believes, on which the distal reward is contingent.

The sensitivity of children with ADHD to delay was normalized when extra stimulation was applied in a delay aversion task (Antrop, et al., 2006). Probably, this was due to the fact that the perceptual resources were allocated away from the frustrating effects of delay resulting in an increased tolerance to delay (Mischel & Baker, 1975; Sonuga-Barke, 1994). Besides this allocation of attention to already existing stimulation, coping with delay aversion can also result in hyperactive behaviour like fidgeting as a mean of stimulation that reduces the awareness of time (Antrop, Buysse, Roeyers, & Van Oost, 2005). The excessive unnecessary mouse responses (CLICKCAT) in our sample may, thus, be interpreted as a form of secondary hyperactivity evolving in delay rich situations (Sonuga-Barke, 2005).

The correct classification of 65% of the ADHD cases and 61% of the controls is satisfactory for a single neuropsychological measure and, particularly, for a measure of motivation. The correct classification improved to 74% of the children with ADHD and 70% of the controls when the information whether a child belonged to the group of extreme CLICK responders or not was added to the predictive model. This finding provides further evidence for the postulated need to combine measures of different domains in neuropsychological assessment of ADHD (Doyle, Biederman, Seidman, Weber, & Faraone, 2000; Nigg, 2005; Solanto, et al., 2001).

In line with findings in other studies of delay choice (Marco, et al., 2008; Olson, et al., 2007), we found a positive effect of IQ on the overall waiting times which, however, disappeared when corrected for condition. Given the inconsistent findings in the literature (Olson, et al., 2007), the small sample size, and the reduced size of the battery, no substantial increase of knowledge regarding this issue is

provided by the present study. Similarly, with respect to the small but also inconsistent gender effects on delay aversion (Silverman, 2003) one should be cautious in generalizing that there are no significant gender effects on ConDAT variables.

Limitations

Even despite the large effect sizes of a delay aversion measure and the good predictive validity of the newly introduced task, there is still a large amount of unexplained variation. The effects of age and also of time-on-task, revealing differential age effects in ADHD children and controls were well captured. However, other factors could not be controlled, e.g., the actual mood state or individual physiological aspects like hunger or tiredness in the children. Also further psychosocial factors were not covered by the design. Thus, whether a child felt comfortable in the actual situation and with the experimenter, or the attraciveness of the activities to be awaited after the assessment may have also influenced the delay choices. These uncontrolled conditions may, so far, limit diagnostic utility of the new task.

Furthermore, most of the children tolerated delay to an extent far away from an optimal reward/delay ratio, indicating that a large part of the motivation for delay choice is not covered by the task. The latter include, for example, personal ambitions and a learned competitive attitude resulting in to get the highest reward that is possible,

The findings about psychopathological effects on ConDAT variables are not discussed in detail here because important questions like stratification effects, e.g. the role of ADHD as mediating factor between comorbid measures and delay measures, cannot be answered with regard to our study design, the relative small sample size, and missing information from parents.

Summary

In conclusion, the present study provides new additional evidence for the role of delay sensitivity as a core characteristic of ADHD. The findings demonstrate a missing adaptation to temporal context in children with ADHD. Obviously, children with ADHD have a history of failing to adapt to situations with low stimulus density especially when delay is involved. This history may have led to an acquired delay aversion that overrules accommodative perceptions and leads to a chronic delay intolerance. The ConDAT task has been shown to be suited for identifying ADHD related motivational deficits and, furthermore, provides a better prediction when the implemented measure of motor hyperactivity is included indicating that there is substantial neuropsychological heterogeneity in ADHD.

4.6 References

Alvarez, J. A., & Emory, E. (2006). Executive function and the frontal lobes: a meta-analytic review. Neuropsychology Review, 16(1), 17-42.

Antrop, I., Stock, P., Verte, S., Wiersema, 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 & Psychiatry & Allied Disciplines, 47(11), 1152-1158.

Biederman, J., & Faraone, S. V. (2005). Attention-deficit hyperactivity disorder. Lancet, 366(9481), 237-248.

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing cognition in ADHD: beyond executive dysfunction. Trends in Cognitive Sciences, 10(3), 117-123.

Castellanos, F. X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: the search for endophenotypes. Nature Reviews Neuroscience, 3(8), 617-628.

Conners, C. K. (1997a). Conners' Parent Rating Scale - Revised (L) / Conners' Teacher Rating Scale - Revised (L). New York: Multi-Health Systems Inc.

Conners, C. K. (1997b). Conners' Teacher Rating Scale - Revised (L). New York: Multi-Health Systems Inc.

Douglas, V. I., & Parry, P. A. (1994). Effects of reward and nonreward on frustration and attention in attention deficit disorder. Journal of Abnormal Child Psychology, 22(3), 281-302.

Doyle, A. E., Faraone, S. V., Seidman, L. J., Willcutt, E. G., Nigg, J. T., Waldman, I. D., et al. (2005). Are endophenotypes based on measures of executive functions useful for molecular genetic studies of ADHD? Journal of Child Psychology & Psychiatry & Allied Disciplines, 46(7), 774-803.

Gaub, M., & Carlson, C. L. (1997). Gender differences in ADHD: a meta-analysis and critical review.[erratum appears in J Am Acad Child Adolesc Psychiatry 1997 Dec;36(12):1783]. Journal of the American Academy of Child & Adolescent Psychiatry, 36(8), 1036-1045.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: a research note. Journal of Child Psychology & Psychiatry & Allied Disciplines, 38(5), 581-586.

Green, L., Fry, A. F., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. Psychological Science, 5(1), 33-36.

Green, L., Myerson, J., Lichtman, D., Rosen, S., & Fry, A. (1996). Temporal discounting in choice between delayed rewards: The role of age and income. Psychology and Aging, 11(1), 79-84.

Haenlein, M., & Caul, W. F. (1987). Attention deficit disorder with hyperactivity: a specific hypothesis of reward dysfunction. Journal of the American Academy of Child & Adolescent Psychiatry, 26(3), 356-362.

Heinrich, H., Moll, G. H., Dickhaus, H., Kolev, V., Yordanova, J., & Rothenberger, A. (2001). Time-on-task analysis using wavelet networks in an event-related potential study on attention-deficit hyperactivity disorder. Clinical Neurophysiology, 112(7), 1280-1287.

Heyder, K., Suchan, B., & Daum, I. (2004). Cortico-subcortical contributions to executive control. Acta Psychologica, 115(2-3), 271-289.

Kuntsi, J., Oosterlaan, J., & Stevenson, J. (2001a). Psychological mechanisms in hyperactivity: I. Response inhibition deficit, working memory impairment, delay aversion, or something else? Journal of Child Psychology & Psychiatry & Allied Disciplines, 42(2), 199-210.

Kuntsi, J., Stevenson, J., Oosterlaan, J., & Sonuga-Barke, E. J. (2001b). Test-retest reliability of a new delay aversion task and executive function measures. British Journal of Developmental Psychology, 19(3), 339-348.

Luman, M., Oosterlaan, J., & Sergeant, J. A. (2005). The impact of reinforcement contingencies on AD/HD: a review and theoretical appraisal.[erratum appears in Clin Psychol Rev. 2005 Jun;25(4):533]. Clinical Psychology Review, 25(2), 183-213.

Marco, R., Miranda, A., Schlotz, W., Melia, A., Mulligan, A., Mueller, U., et al. (2008). Delay and Reward Choice in ADHD: An Experimental Test of the Role of Delay Aversion. Neuropsychology, Manuscript in-press.

McClure, S. M., York, M. K., & Montague, P. R. (2004). The neural substrates of reward processing in humans: the modern role of FMRI. Neuroscientist, 10(3), 260-268.

Mischel, W., & Ayduk, O. (2004). Willpower in a cognitive-affective processing system: The dynamics of delay of gratification. In R. F. Baumeister & K. D. Vohs (Eds.), Handbook of self-regulation: Research, theory, and applications (pp. 99-129). New York, NY: Guilford Press.

Müller, U. C., Sonuga-Barke, E., & Steinhausen, H. C. (2009). Willingness to Wait for Rewards in Childhood and Adolescence in the Face of Diminishing Returns. Manuscript submitted for publication.

Müller, U. C., Sonuga-Barke, E. J., Brandeis, D., & Steinhausen, H.-C. (2006). Online measurement of motivational processes: introducing the Continuous Delay Aversion Test (ConDAT). Journal of Neuroscience Methods, 151(1), 45-51.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? Psychological Bulletin, 127(5), 571-598.

Nigg, J. T., Goldsmith, H. H., & Sachek, J. (2004). Temperament and attention deficit hyperactivity disorder: the development of a multiple pathway model.[see comment]. Journal of Clinical Child & Adolescent Psychology, 33(1), 42-53.

Nigg, J. T., Willcutt, E. G., Doyle, A. E., & Sonuga-Barke, E. J. (2005). Causal heterogeneity in attention-deficit/hyperactivity disorder: do we need neuropsychologically impaired subtypes? Biological Psychiatry, 57(11), 1224-1230.

Olson, E. A., Hooper, C. J., Collins, P., & Luciana, M. (2007). Adolescents' performance on delay and probability discounting tasks: Contributions of age, intelligence, executive functioning, and self-reported externalizing behavior. Personality and Individual Differences, 43(7), 1886-1897.

Petry, N. M. (2002). Discounting of delayed rewards in substance abusers: relationship to antisocial personality disorder. Psychopharmacology, 162(4), 425-432.

Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: a systematic review and metaregression analysis.[see comment]. American Journal of Psychiatry, 164(6), 942-948.

Rapport, M. D., Tucker, S. B., DuPaul, G. J., Merlo, M., & Stoner, G. (1986). Hyperactivity and frustration: the influence of control over and size of rewards in delaying gratification. Journal of Abnormal Child Psychology, 14(2), 191-204.

Reynolds, B., Richards, J. B., Horn, K., & Karraker, K. (2004). Delay discounting and probability discounting as related to cigarette smoking status in adults. Behavioural Processes, 65(1), 35-42.

Sagvolden, T., Aase, H., Zeiner, P., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. Behavioural Brain Research, 94(1), 61-71.

Sagvolden, T., & Sergeant, J. A. (1998). Attention deficit/hyperactivity disorder--from brain dysfunctions to behaviour. Behavioural Brain Research, 94(1), 1-10.

Sattler, J. M. (1992). Assessment of Children: WISC III and WPSSI-R Supplement. Sandiego, CA: M. Sattler.

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(11), 2092-2103.

Seidel, W. T., & Joschko, M. (1990). Evidence of difficulties in sustained attention in children with ADDH. Journal of Abnormal Child Psychology, 18(2), 217-229.

Sergeant, J. (2000). The cognitive-energetic model: an empirical approach to attention-deficit hyperactivity disorder. Neuroscience & Biobehavioral Reviews, 24(1), 7-12.

Sergeant, J. A. (2005). Modeling attention-deficit/hyperactivity disorder: a critical appraisal of the cognitive-energetic model. Biological Psychiatry, 57(11), 1248-1255.

Silverman, I. W. (2003). Gender Differences in Delay of Gratification: A Meta-Analysis. [Journal Peer Reviewed Journal]. Sex Roles, 49(9-10), 451-463.

Sonuga-Barke, E. J. (2002). Psychological heterogeneity in AD/HD--a dual pathway model of behaviour and cognition. Behavioural Brain Research, 130(1-2), 29-36.

Sonuga-Barke, E. J. (2005). Causal models of attention-deficit/hyperactivity disorder: from common simple deficits to multiple developmental pathways. Biological Psychiatry, 57(11), 1231-1238.

Sonuga-Barke, E. J., Sergeant, J. A., Nigg, J., & Willcutt, E. (2008). Executive dysfunction and delay aversion in attention deficit hyperactivity disorder: nosologic and diagnostic implications. Child & Adolescent Psychiatric Clinics of North America, 17(2), 367-384.

Sonuga-Barke, E. J., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion--I. The effect of delay on choice. Journal of Child Psychology & Psychiatry & Allied Disciplines, 33(2), 387-398.

Spencer, T. (2006). ADHD and comorbidity in childhood. Journal of Clinical Psychiatry, 8, 27-31.

Spencer, T., Biederman, J., & Mick, E. (2007). Attention-deficit/hyperactivity disorder: diagnosis, lifespan, comorbidities, and neurobiology. Journal of Pediatric Psychology, 32(6), 631-642.

SPSS for Windows, Rel. 17.0.0, (2008). Chicago: SPSS Inc.

Tewes, U., Rossmann, P., & Schallberger, U. (2000). Hamburg-Wechsler-Intelligenztest für Kinder. Göttingen: Huber.

Walls, R. T. (1973). Delay of reinforcement development. Child Development, 44(3), 689-692.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review. Biological Psychiatry, 57(11), 1336-1346.

5 Allgemeine Diskussion

Die vorliegende Arbeit will als Ganzes einen Beitrag leisten zum besseren Verständnis motivationaler Ursachen der Aufmerksamkeitsdefizit- / Hyperaktivitätsstörung ADHS, besonders der mangelnden Fähigkeit von Kindern mit ADHS, aus eigenem Antrieb längerfristige Ziele zu verfolgen. Dieses als Delay Aversion (Sonuga-Barke, Taylor, Sembi, & Smith, 1992) bezeichnete Phänomen ist allerdings aus verschiedenen Gründen mit neuropsychologischen Methoden schwierig zu erforschen. Der wichtigste Grund dafür ist die Tatsache, dass sich die Fähigkeiten, auf kurzfristige kleine Belohnungen angesichts längerfristiger Ziele zu verzichten, mit dem Älterwerden stark entwickeln. Dies hat zur Folge, dass die meisten der bisher angewandten neuropsychologischen Tests bezüglich der Reichweite und Sensitivität ihres Messbereichs an Grenzen stossen, wenn es darum geht, mit dem gleichen Mass sowohl jüngere Kinder als auch Jugendliche oder Erwachsene valide zu erfassen und zu vergleichen (z.B. Marco, et al., 2008).

5.1 Zu den Zielen dieser Arbeit

Das wichtigste Ziel dieser Arbeit war die Entwicklung eines Tests, welcher diese und andere methodische Grenzen überwindet und modernen Anforderungen an neuropsychologische Instrumente genügt, die im Bereich der Neurowissenschaften und allenfalls der Genetik eingesetzt werden können (Alvarez & Emory, 2006; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Castellanos & Tannock, 2002). Die bedeutendste Herausforderung war es, eine Testanlage zu finden, welche es erlaubt, die Entscheidung zwischen einer kleinen unmittelbaren Belohnung und einer grösseren verzögerten sowohl kleinen Kinder als auch erwachsenen Personen anzubieten und in einem einheitlichen Mass erfassen. Es stellte sich die Frage nach der Art und Grösse der Belohnungen sowie nach den Grössen- und Zeitverhältnissen zwischen der unmittelbaren und der verzögerten Belohnung.

Zudem mussten folgende Bedingungen erfüllt sein: Der ganze Prozess, einschliesslich der Auslieferung der verzögerten Belohnung, sollte möglichst direkt erfasst und in den Testablauf integriert sein.

Der Test sollte möglichst alles Verhalten, welches die Abneigung gegen das Warten auf Belohnung (worunter auch das Entlassung aus einer unangenehmen Situation verstanden werden kann) positiv oder negativ beeinflusst, erfassen, also z.B. verhindern, dass die Testpersonen während des Wartens durch Beschäftigungen mit ablenkenden Tätigkeiten ihre negativen Empfindungen reduziert und dadurch ihre Fähigkeiten zum Belohnungsaufschub überschätzt werden. Schliesslich sollte der Test auch für Messungen im EEG geeignet sein.

5.2 Zur Entwicklung des ConDAT Tests

In der Physik gelangen Skalen zur Anwendung, welche die Messung eines Phänomen einerseits über einen grossen Messbereich hinweg und, andererseits mit ausreichender Differenzierung in jedem Bereich erlauben; ein Beispiel ist die logarithmischen Dezibel-Skala. Aus derartigen Skalen wurde die Grundidee für ein Verfahren mit einer kontinuierlichen und sich stetig verringernden Belohnungsausschüttung abgeleitet - im Gegensatz zu den bisher meistens angewandten Verfahren, bei denen im Voraus Entscheidungen zwischen zwei Ereignissen verschiedener Grösse zu verschiedenen Zeitpunkten zu fällen sind (z.B. Green, Myerson, & Ostaszewski, 1999; Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001; Marco, et al., 2008; Scheres, et al., 2006). An die Stelle der Entscheidung, entweder jetzt wenig oder später viel zu erhalten, war somit die Frage getreten, wie viel Verzögerung angesichts einer sich stetig verringernden Rendite in Kauf genommen wird, um die Belohnung zu erhöhen.

Eine weitere zentrale Idee war der Ersatz von materiellen Belohnungen, welche bekanntlich stark alters- und einkommensabhängig sind, durch eine Anzahl von ideellen Belohnungen, welche von der Testperson ausgewählt werden können. Damit sollten Alters- und Geschlechtsunterschiede, aber auch ökonomische Faktoren in der Valenz von Belohnungen möglichst reduziert werden (Green, Myerson, Lichtman, Rosen, & Fry, 1996; Silverman, 2003). Die vier zur Verfügung stehenden kurzen Videoclips, von denen einer als Belohnung im Anschluss an den letzten Testdurchgang in der Länge gemäss der gesammelten symbolischen Belohnung gezeigt wird, sind nach dem Kriterium ausgewählt, möglichst mindestens ein interessantes Thema für jedes Alter und beide Geschlechter zu behandeln.

In ersten Vorversuchen hatte sich allerdings gezeigt, dass der Test immer noch zu Deckeneffekten führte, wenn in den bisherigen Tests angewandte Zeit- und Grössenverhältnisse zwischen den beiden Belohnungstypen in das kontinuierliche Konzept übernommen wurden. Dies führte zur Weiterentwicklung des Verfahrens einerseits durch die Einführung von Testdurchgängen mit offenem Ende, welche nur durch eine aktive Entscheidung der Testperson enden können, also Deckeneffekte praktisch ausschliessen. Andererseits durch Veränderung der Belohnungskurve in Richtung einer sich schneller verringernden Belohnungsrate, welche auch bei sehr geduldigen Personen einen Abbruch der Durchgänge provoziert.

Durch den Einbau von gelegentlichen Unterbrechungen des Belohnungsmechanismus, welcher nur durch die Testperson wieder aktiviert werden kann, wurde einerseits ein Mittel eingebaut, die so genannte Off-task-Aktivität zu verringern, andererseits ein Parameter eingeführt, welcher – im Gegesatz zur Aktion zum Beenden des Durchganges – positiv motiviert ist und das Verlangen nach mehr Belohnung repräsentiert. Die diesen beiden Kommandos entsprechenden Tastenimpulse können an ein EEG-Aufzeichnungsgerät übermittelt werden und erlauben einen Vergleich zwischen den Abschnitten, welche den positiven und den negativen Antworten vorausgehen, beispielsweise hinsichtlich der dominierenden Frequenzen.

5.3 Zu den Testgütekriterien

Gemäss den Annahmen der Testtheorie muss ein Test hauptsächlich über zwei Qualitäten verfügen: über eine gute Messgenauigkeit oder Reliabilität und über eine genaue Vorhersagefähigkeit oder Validität (Fischer, 1968). Die Test-Retest-Reliabilität des ConDAT wurde in Studie 1 untersucht und liegt mit r=.06 in einem Bereich, welcher für psychologische Tests als ausreichend angesehen werden kann, wenn man die Komplexität und Situationsabhängigkeit des Konstruktes sowie das grosse Intervall von 12 Wochen zwischen den Testungen berücksichtigt (Kuntsi, et al., 2001).

Studie 3 hat die prädiktive Validität als Form der Konstruktvalidität des Tests untersucht. Die Hauptfrage, ob der Test misst, was er zu messen vorgibt (Konstruktvalidität), d.h., ob er tatsächlich ein Mass

für Delay Aversion ist, wurde indirekt überprüft, indem seine Fähigkeit untersucht wurde, eine Person einer von zwei Gruppen zuzuordnen, welche sich gemäss dem aktuellen Stand des Wissens unter anderem in ihrer Neigung zu Delay Aversion unterscheiden. Die Fähigkeit des ConDAT, eine Person korrekt entweder der ADHS-Gruppe, von welcher eine abnormale Tendenz zu Delay Aversion angenommen wird, oder der Kontrollgruppe ohne eine solche Tendenz zuzuordnen kann mit 72% als überraschend gut im Vergleich mit eher kognitiv ausgerichteten neuropsychologischen Tests (Doyle, Biederman, Seidman, Weber, & Faraone, 2000) bezeichnet werden. Allerdings bezieht diese Vorhersage auch noch ein Hyperaktivitätsmass mit ein, weswegen streng genommen nicht von Konstruktvalidität bezüglich Delay Aversion gesprochen werden kann. Selbst wenn nur das Ausmass des mittleren tolerierten Belohnungsaufschubs, also ein ‚reines' Mass für Belohnungsaufschub, zur Vorhersage benutzt wird, werden immer noch 63% der Probanden der richtigen Gruppe zugeordnet. Enstsprechend ist die Effektstärke dieses Masses von 0.8 als hoch einzustufen (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

5.4 Zu den inhaltlichen Befunden

Nach Kenntnis des Verfassers konnte erstmals die Entwicklung von Delay Aversion über die gesamte Altersspanne von 6 bis 17 Jahren mittels eines geeigneten neuropsychologischen Verfahrens dargestellt werden (Studie 2). Die Resultate bestätigen die auf anderen Befunden beruhenden Hypothesen, wonach die Fähigkeit zu Belohungsaufschub mit dem Alter zunimmt (Green, Fry, & Myerson, 1994; Marco, et al., 2008; Walls, 1973). Es konnte kein Hinweis dafür gefunden werden, dass die Art der Belohnung mit dem Alter in bedeutendem Ausmass abnimmt, was sehr wahrscheinlich mit dem Konzept einer ideellen adaptiven Belohnung zusammenhängt.

Es konnte weiterhin gezeigt werden, dass nicht nur die allgemeine Fähigkeit zum Belohnungsaufschub mit dem Alter zunimmt, sondern auch – und statistisch unabhängig davon – die Fähigkeit zunimmt, ein gewähltes Ziel über den Zeitraum einer Testsituation konsequent durchzuhalten, welche

mit den vorwiegend in der Adoleszenz sich entwickelnden Fähigkeit zur Selbstregulation im Sinne von Exekutivfunktionen in Verbindung gebracht werden kann (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; McCormack, Brown, Maylor, Darby, & Green, 1999). Konzeptionell haben wir zwischen einer vom zeitlichen Kontext unabhängigen und einer abhängigen Komponente von Delay Aversion gesprochen. Diese Unterscheidung hat sich in Studie 3 als äusserst wichtig und hilfreich erwiesen. Entgegen der Erwartung gab es bei den Kindern mit ADHS bezüglich keiner der Komponenten, also weder bezüglich der mittleren Wartezeit noch bezüglich des Verlaufs der Wartezeiten, Alterseffekte.

Die ADHS-Gruppe investierte insgesamt weniger Zeit in das Sammeln der Belohnung und zeigte vom Beginn des Tests bis zum Ende einen gleich bleibenden Verlauf der Wartezeiten. Obwohl wir bei gesunden Kindern eher gleich bleibende oder steigende Wartezeiten im Verlauf des Tests als Zeichen entwickelter Exekutivfunktionen interpretiert haben, widerstrebt es uns nicht nur intuitiv, hier besser entwickelte Exekutivfunktionen zu vermuten, sondern auch im Hinblick auf die vielfach berichteten Befunde über ein exekutives Defizit bei ADHS (Barkley, 1997; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Willcutt, et al., 2005).

Dagegen bietet das erweiterte Dual Pathway-Modell (Sonuga-Barke, 2005) einen plausiblen Erklärungsansatz: Der Begriff Delay Tolerance steht für die unpathologische und situationsabhängige Fähigkeit, in der Zukunft liegende Ziele zu verfolgen, welche sich mit wachsendem Alter verbessert. Dem gegenüber wird unter Delay Aversion eine generalisierte emotional negativ gefärbte Haltung gegenüber Situationen, welche Durchhaltevermögen verlangen, verstanden. Unsere Befunde stimmen insofern mit diesem Modell überein, als sowohl die kürzeren Wartezeiten als auch das Fehlen der bei jungen Kontrollkindern eher überschiessenden Motivation zu Beginn der Aufgabe vereinbar sind mit der Hypothese einer chronischen, generalisierten, und nicht adaptiven Abneigung, zu Gunsten längerfristigen Ziele auf kurzfristige Belohnungen zu verzichten, kurz: mit Delay Aversion.

5.5 Kritische Anmerkungen

Die wichtigste Kritik erwächst aus der grossen Varianz der ermittelten Befunde, welche sich nicht auf die gemessenen Einflussgrössen zurückführen lassen. Auf Grund dieser Fehlervarianz liegt auch dieses Verfahren im Verbund mit fast allen neuropsychologischen Einzeltests (Willcutt, et al., 2005) nicht im Bereich des individuellen diagnostischen Einsatzes. Vermutlich spielen State-Faktoren eine grössere Rolle, als dies bei eher kognitiven Leistungstests der Fall ist. Die Motivation, auf einen kleinen Filmausschnitt zu warten, hängt sehr stark von situativen Gegebenheiten ab: von der momentanen Stimmung des Probanden, von physiologischen Zuständen, welche die Wachheit beeinflussen, von der Sympathie für die Versuchsleiter, vom Gesamtkontext einschliesslich der nach dem Test geplanten Tätigkeit etc. Es wäre deshalb sicher wünschenswert, solche situationsgebundenen Faktoren zum Zeitpunkt der Messung zu erfassen.

Ein weiteres interessantes Phänomen ist die Tatsache, dass die meisten Kinder viel mehr an Belohnung sammelten, als für das Betrachten des fast vollständigen Filmausschnitts nötig gewesen wäre. Die Kinder der gesunden Kontrollstichprobe setzten durchschnittlich die Häfte ihrer investierten Wartezeit für 99.3% des Filmausschnittes ein, während die andere Hälfte für zusätzliche 0.1% eingesetzt wurden. Diese eindrückliche Ineffizienz ist zu einem Teil verursacht durch die in der Testkonstruktion verfolgten Strategie, das Ausschütten der Belohnung früh und stark zu reduzieren, damit die meisten Personen den Test, dessen Dauer offen ist, innerhalb einer akzeptablen Zeit beenden.

Ein grosser Teil der Motivation muss demnach aus Quellen stammen, welch nicht mit der explizit ausgesetzten Belohnung im Zusammenhang stehen, sondern insofern als intrinsisch bezeichnet werden können, als sie weder durch den Test selbst noch durch eine Anweisung des Testleiters hervorgerufen werden. Die Vermutung liegt nahe, dass hier auch internalisierte Leistungsmotivation eine Rolle spielt, welche es wahrscheinlich erstrebenswert macht, in diesem Test sogar ohne Belohnung eine gewisse Zeit durchzuhalten, d.h. das Verhalten zu maximieren, anstatt, wie instruiert, zu optimieren. Wir müssen folgerichtig auch annehmen, dass ein wesentlicher Teil der Belohnung darin bestand, aus einer zunehmend negativen Situation befreit zu werden.

5.6 Ausblick

Die vorliegenden Untersuchungen haben insgesamt nicht nur ein neues Instrument zur neuropsychologischen Messung der Fähigkeit, längerfristige Ziele zu verfolgen, eingeführt und etabliert, sondern auch differenzierte Erkenntnisse über deren Entwicklung, Beschaffenheit und Veränderung unter ADHS erbracht.

Für weiterührende Studien kann der ConDAT test hinsichtlich seiner grosse Erfassungsspannweite, seiner differenzierten Ausgangsparameter, sowie des nicht-materiellen Belohnungskonzeptes Vorbild sein. Eine starke Verbesserung hingegen würde es bedeuten, wenn die Belohnung in grösserem Masse zur Varianz der Bereitschaft, zu warten, beitragen würde, als dies beim ConDAT zur Zeit noch der Fall ist. Eine Möglichkeit und zugleich eine grosse Herausforderung bestünde darin, die Belohnung so zu erhöhen, dass sie im Verhältnis zur intrinsischen Motivation stärkeren Einfluss auf das Testverhalten gewinnt, ohne jedoch eine solche Verbesserung des ‚Wirkungsgrades' mit dem ökonomischen Preis einer längeren Testdauer zu bezahlen. Der gegenteilige Weg würde bedeuten, dass die tatsächliche Motivation besser erforscht wird und erfasst werden kann. Ein erster Ansatz in diese Richtung könnte ein Delay Aversion Test vollkommen ohne explizite Belohnung sein. Die stärkere Berücksichtigung von impliziten Belohnungen bzw. intrinsischer Motivation würde zudem wahrscheinlich die ökologische Validität eines solchen Tests erhöhen, jedenfalls im Hinblick auf den Schulalltag von Kindern mit ADHS.

Die wünschenswerte Weiterentwicklung in Richtung eines valideren Paradigmas zur Messung der Fähigkeit zur Selbstregulierung stünde jedoch nicht nur im Dienste weiterer Erkenntnisse über motivationale Defizite bei ADHS, sondern auch der Untersuchung normaler Aspekte der Selbstregulation oder deren pathologischen Veränderung bei anderen Krankheitsbildern, beispielsweise Suchtkrankheiten.

Schliesslich wäre zu wünschen, dass die im ConDAT angelegte Möglichkeit, Hirnprozesse im Zusammenhang mit Delay Aversion zu verfolgen, genutzt wird und weitere Erkenntnisse über die Pro-

zesse erbringen kann, welche der Entscheidung zu Grunde liegen, ein anvisiertes Ziel entweder weiter zu verfolgen oder angesichts unmittelbarer Anreize aufzugeben.

5.7 Literatur

Alvarez, J. A., & Emory, E. (2006). Executive function and the frontal lobes: a meta-analytic review. *Neuropsychology Review, 16*(1), 17-42.

Anderson, V. A., Anderson, P., Northam, E., Jacobs, R., & Catroppa, C. (2001). Development of executive functions through late childhood and adolescence in an Australian sample. *Developmental Neuropsychology, 20*(1), 385-406.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing cognition in ADHD: beyond executive dysfunction. *Trends in Cognitive Sciences, 10*(3), 117-123.

Castellanos, F. X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: the search for endophenotypes. *Nature Reviews Neuroscience, 3*(8), 617-628.

Doyle, A. E., Biederman, J., Seidman, L. J., Weber, W., & Faraone, S. V. (2000). Diagnostic efficiency of neuropsychological test scores for discriminating boys with and without attention deficit-hyperactivity disorder. *Journal of Consulting & Clinical Psychology, 68*(3), 477-488.

Fischer, G. H. (1968). *Einführung in die Theorie Psychologischer Tests*. Bern: Hans Huber.

Green, L., Fry, A. F., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. *Psychological Science, 5*(1), 33-36.

Green, L., Myerson, J., Lichtman, D., Rosen, S., & Fry, A. (1996). Temporal discounting in choice between delayed rewards: The role of age and income. *Psychology and Aging, 11*(1), 79-84.

Green, L., Myerson, J., & Ostaszewski, P. (1999). Discounting of delayed rewards across the life span: Age differences in individual discounting functions. *Behavioural Processes, 46*(1), 89-96.

Kuntsi, J., Stevenson, J., Oosterlaan, J., & Sonuga-Barke, E. J. (2001). Test-retest reliability of a new delay aversion task and executive function measures. *British Journal of Developmental Psychology, 19*(3), 339-348.

Marco, R., Miranda, A., Schlotz, W., Melia, A., Mulligan, A., Mueller, U., et al. (2008). Delay and Reward Choice in ADHD: An Experimental Test of the Role of Delay Aversion. *Neuropsychology*, Manuscript in-press.

McCormack, T., Brown, G. D., Maylor, E. A., Darby, R. J., & Green, D. (1999). Developmental changes in time estimation: comparing childhood and old age. *Developmental Psychology, 35*(4), 1143-1155.

Nigg, J. T., Willcutt, E. G., Doyle, A. E., & Sonuga-Barke, E. J. (2005). Causal heterogeneity in attention-deficit/hyperactivity disorder: do we need neuropsychologically impaired subtypes? *Biological Psychiatry, 57*(11), 1224-1230.

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*(11), 2092-2103.

Silverman, I. W. (2003). Gender Differences in Delay of Gratification: A Meta-Analysis. *Sex Roles, 49*(9-10), 451-463.

Sonuga-Barke, E. J. (2005). Causal models of attention-deficit/hyperactivity disorder: from common simple deficits to multiple developmental pathways. *Biological Psychiatry, 57*(11), 1231-1238.

Sonuga-Barke, E. J., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion--I. The effect of delay on choice. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 33*(2), 387-398.

Walls, R. T. (1973). Delay of reinforcement development. *Child Development, 44*(3), 689-692.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review. *Biological Psychiatry, 57*(11), 1336-1346.

6 Appendix

6.1 Description of the ConDAT task

One special characteristic of the ConDAT task is its adaptive non-material reward concept, realized by a video clip, which can be chosen among four clips covering different interests (e.g. sports, animals, culture). A part of the chosen clip is automatically played after the task, whereas the duration of the played part, which can be two minutes at maximum, represents the amount of reward. The delay of the reward delivery obviously is identical to the time on task due to the immediate delivery of the reward after the last trial has finished.

In contrast, the amount of the reward is linked to the time on task (and thus, to the delay), by a more complex mechanism which a) uses a token to visualize the rewarding process to the proband, and b) models the antinomy between an immediate small reward and a delayed large reward in a continuous way. Identically in all of the 30 trials, the token is delivered continuously, but in a permanently decreasing magnitude. For each time point of the trial the token delivery is calculated by the equation (1)

$$rew = -\log\left(\frac{fr}{c^2 + fr}\right)$$

where rew denotes the amount of the token, fr the elapsed time in “frames”, i.e. in units of about 1/10 sec., and c a constant. This function leads to a large amount of token delivery at the beginning of each trial, which steadily diminishes and asymptotically reaches zero after a certain time (see Figure 1).

Because the duration of the video clip is calculated from the average of the collected token in all of the 30 trials, it is, theoretically, only played in the maximum possible duration of two minutes, if a person collects the maximum possible token in each trial, i.e. if he collects the token until the function reaches zero, which is the case after 60 seconds (600 frames). However, if one is waiting only 50% of the maximal time, i.e. 30 seconds each trial, only ½ second of the clip “gets lost”. As the curve’s gradient

in Figure 1 shows, collecting the token is strongly rewarded only at the very beginning of a trial, which leads to an unfavourable time/reward ratio very soon. The token delivery is visualized as gold passing from a donkey's mouth into a container and exactly follows the mathematical reward function both in the amount of gold passing from the donkey's mouth and in the increase of gold in the container. As the black squares at the bottom of Figure 1 symbolize, the gold stream is steadily at the beginning of each trial, becomes then interrupted more and more, until only small drops in large intervals. By this visualization, the proband permanently is aware of the actual benefit resulting from waiting, in other words, of the actual benefit resulting from accepting delay.

The mission of the proband is now simply to terminate a trial by pressing the spacebar, when he judges the amount of reward not worth waiting (and therefore adding delay) any more. The trial time therefore reflects the critical transition from tolerating delay to dismissing delay. In comprehensive pilot tests, the parameters of the rewarding function have been optimized in order to capture this critical transmission in subjects of different age, gender, and psychopathology.

As an additional feature of the task, the rewarding process is completely interrupted five times in each trial, indicated by the stopped gold flow and an appearing question mark on the screen. The rewarding process can be reactivated by pressing a mouse button or, alternatively, the trial can be terminated by pressing the space bar. These stops are implemented for two reasons: First, to provide triggers related to positive responses (wanting more reward), which can be used in recordings of evoked potentials, and which can be contrasted to potentials related to negative responses associated with quitting a trial by pressing the spacebar. Second, to keep the proband involved in the task and minimize uncontrolled off-task activity which probably would bias the tolerance or aversion to delay.

After each trial, an information screen is displayed indicating how many of the 30 trials remain by showing the according number of donkeys, and how much of the clip is played, no change of the collecting strategy assumed, represented by a film strip of the corresponding length. Mathematically, the length of the film strip corresponds to the average of the so far collected token across all trials.

6.2 Example data

Table A1: Output measures of the ConDAT task

	Score / category	Variable
Gender	male	
Group	controls	
Age	8.2 years	
Mean rewarded time / trial	24.62 seconds	CT
Mean change of rewarded time / trial (slope of regression line)	-1.29 seconds	PERSIST
Unnecessary mouse responses	10	CLICK
Group of mouse responders	low	CLICKCAT

Table A2: Relations between actual time, rewarded time, reward delay, and reward size

Measure	seconds	minutes	% of max.	% of total time on task
Rewarded time on task				
Total rewarded time	739	12.3	41.0%	69.3%
Mean rewarded time / trial (DELAY)	25	0.41	41.0%	
Resulting reward (clip duration) #	119.3	1.99	99.4%	
Total time on task (clock time)				
Total time (= actual delay)	1066	17.8		
Non-rewarded time on task				
Duration of Feedback trials (constant)	1350	135	2.3	12.7%
Total response decision time (varying)	1930	193	3.2	18.1%

Note: # See Appendix I for formula

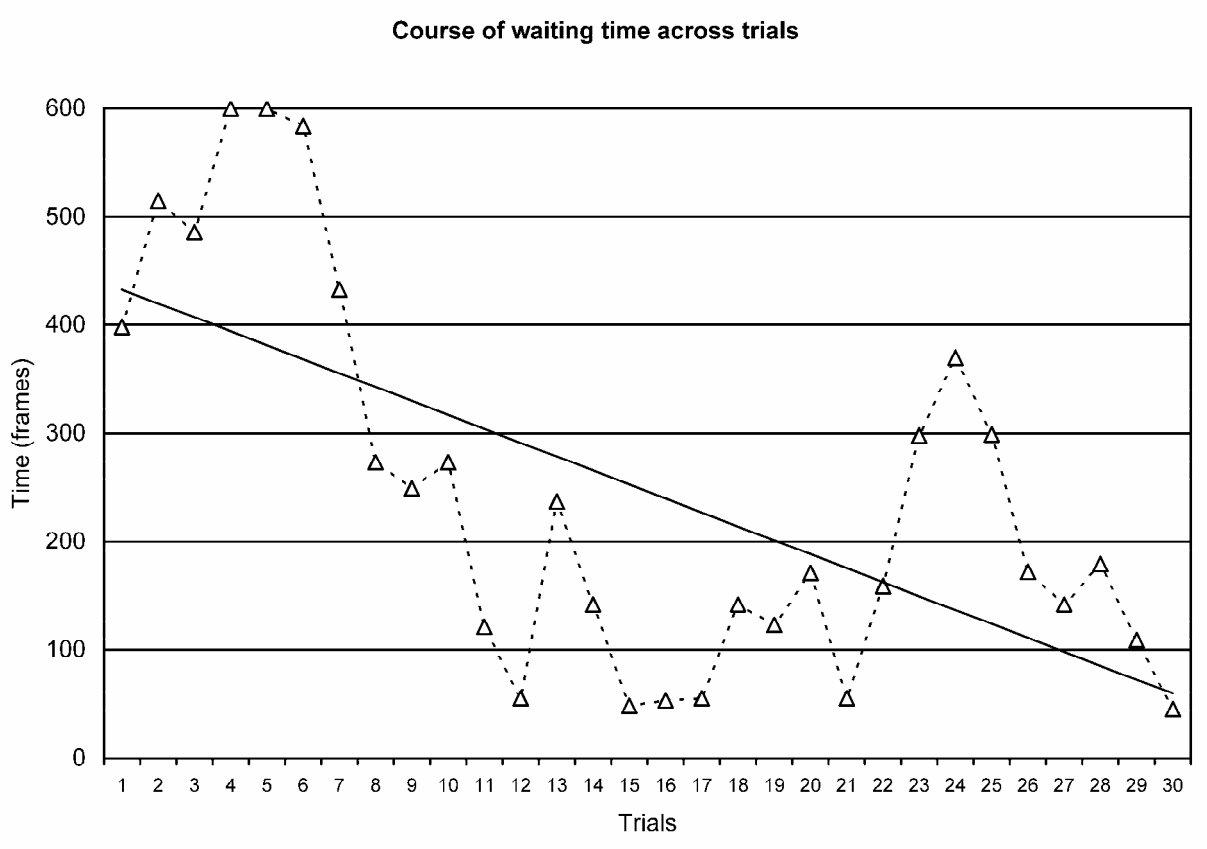

Figure A1: Chart of the example data. The slope of the regression line corresponds to the variable SLOPE. The y-score of the regression line after the first 15 trials, corresponding to the variable DELAY, indicates the mean invested time for collecting reward (indicated in units of "frames", i.e. 1/10 seconds).

6.3 Tables of Study 2

(see next page)

Table A3
Questionnaire and IQ findings

School class	**A**	**B**	**C**	**D**	**E**	**F**	**G**	**H**	**I**	**All**	**Class effects**	
	Mean(SD)	*Mean(SD)*	*Mean(SD)*	*Mean(SD)*	*Mean(SD)*	*Mean(SD)*	*Mean(SD)*	*Mean(SD)*	*Mean(SD)*	*Mean(SD)*	χ^2	*p*
Conners Teacher Rating Scale												
A. Oppositional	62.0 (16.1)	52.8 (13.0)	47.3 (4.4)	52.9 (11.0)	50.1 (6.7)	56.2 (10.9)	49.6 (8.8)	55.5 (12.2)	46.9 (5.4)	52.4 (11.0)	26.8	0.001
B. Cognitive Problems	55.2 (11.3)	50.8 (10.5)	45.3 (3.8)	53.3 (9.4)	51.5 (8.0)	54.5 (8.8)	55.4 (7.3)	56.4 (8.5)	51.8 (9.1)	52.4 (9.1)	34.8	<0.001
C. Hyperactivity	54.8 (10.3)	50.6 (7.9)	46.6 (4.7)	54.1 (12.0)	47.0 (3.7)	54.0 (12.9)	52.6 (10.9)	52.0 (8.8)	45.6 (0.5)	50.7 (9.2)	26.3	0.001
D. Axious-Shy	55.8 (8.7)	56.0 (9.3)	55.3 (9.5)	59.1 (13.7)	53.6 (6.8)	60.8 (9.3)	58.8 (10.5)	62.4 (9.9)	52.4 (9.7)	57.2 (10.1)	18.9	0.016
E. Perfectionism	53.5 (7.7)	54.4 (8.6)	54.6 (9.2)	61.5 (7.2)	49.6 (6.2)	56.3 (7.8)	55.6 (6.9)	52.8 (7.3)	51.4 (5.8)	54.4 (8.0)	28.0	<0.001
F. Social Problems	63.5 (10.0)	50.7 (8.4)	47.2 (3.0)	54.5 (14.3)	48.7 (6.3)	51.3 (6.9)	51.5 (10.8)	50.8 (6.5)	46.4 (1.5)	51.3 (9.2)	35.1	<0.001
H. ADHD Index	57.5 (12.3)	50.7 (8.1)	45.3 (3.9)	52.5 (11.0)	47.0 (5.7)	53.4 (12.8)	53.3 (11.3)	52.0 (10.0)	44.8 (3.5)	50.5 (9.8)	36.5	<0.001
I. GI: Restless-Impulsive	56.9 (11.5)	50.6 (8.0)	46.0 (5.4)	51.6 (10.0)	46.6 (5.6)	54.6 (14.9)	53.5 (11.3)	52.8 (10.1)	45.3 (5.7)	50.7 (10.1)	28.1	<0.001
J. GI: Emotinal Lability	57.8 (12.4)	52.4 (13.3)	49.0 (7.0)	56.5 (14.0)	48.4 (5.5)	56.3 (12.8)	51.1 (9.9)	50.1 (7.1)	45.6 (2.3)	51.8 (10.6)	22.7	0.004
K. GI: Total	58.4 (12.7)	51.0 (9.7)	46.6 (5.5)	53.6 (11.1)	46.9 (5.6)	55.7 (14.7)	53.1 (10.3)	52.3 (8.8)	45.1 (4.1)	51.3 (10.3)	30.7	<0.001
L. DSM: Inattentive	57.1 (13.1)	50.1 (8.2)	44.8 (3.6)	51.7 (9.3)	49.5 (7.5)	52.8 (9.5)	54.3 (7.8)	52.6 (10.2)	46.6 (7.2)	50.8 (9.2)	33.8	<0.001
M. DSM: Hyperactive/Impulsive	55.6 (11.8)	51.0 (8.7)	45.3 (3.4)	53.5 (13.2)	46.7 (4.6)	51.7 (12.3)	51.3 (10.6)	49.3 (9.8)	45.3 (2.6)	49.8 (9.6)	22.2	0.004
N. DSM: Total	56.9 (10.4)	50.6 (7.8)	44.5 (3.1)	52.7 (10.0)	48.0 (6.5)	52.6 (10.7)	53.7 (9.2)	51.6 (10.3)	45.7 (5.1)	50.5 (9.0)	40.7	<0.001
Overall mean	57.3 (11.4)	51.7 (9.4)	47.5 (5.1)	54.4 (11.2)	48.7 (6.0)	54.6 (11.1)	53.3 (9.7)	53.1 (9.2)	47.1 (4.8)	51.8 (9.6)	50.0	<0.001
Strength and Difficulties Questionnaire												
Emotional Symptoms	3.12 (3.37)	1.65 (2.06)	2.15 (1.52)	1.68 (2.21)	0.95 (1.36)	3.16 (2.75)	1.84 (1.83)	2.19 (2.16)	1.06 (2.41)	1.98 (2.27)	19.9	0.011
Conduct Problems	2.88 (2.00)	1.43 (2.46)	0.62 (1.42)	1.21 (1.47)	0.40 (.75)	1.95 (1.90)	0.63 (1.61)	1.38 (1.36)	0.25 (.68)	1.17 (1.76)	43.7	<0.001
Hyperactivity	4.47 (3.41)	3.78 (2.81)	1.73 (2.15)	3.37 (3.40)	1.95 (2.19)	3.58 (3.24)	3.10 (2.36)	3.24 (2.51)	0.75 (1.18)	2.88 (2.81)	28.6	<0.001
Peer Problems	4.29 (2.87)	1.57 (1.78)	0.88 (1.14)	1.63 (2.34)	0.95 (1.70)	1.21 (1.62)	2.40 (2.76)	1.14 (1.11)	0.56 (1.09)	1.58 (2.11)	30.1	<0.001
Prosocial Behaviour	4.00 (2.21)	6.91 (2.07)	9.42 (.76)	7.11 (2.47)	8.55 (2.37)	7.37 (2.22)	5.19 (2.32)	6.14 (1.68)	8.63 (1.67)	7.16 (2.54)	69.9	<0.001
Total Problem score	14.76 (6.12)	8.43 (6.73)	5.38 (4.24)	7.95 (5.46)	4.25 (4.33)	9.89 (7.62)	8.70 (6.73)	7.95 (3.99)	2.63 (2.99)	7.69 (6.26)	44.3	<0.001
Global ratings												
Ambition	3.18 (1.19)	3.26 (0.54)	4.38 (0.64)	4.21 (0.79)	3.20 (0.83)	3.10 (0.97)	2.95 (0.92)	3.24 (1.04)	3.62 (0.72)	3.48 (0.98)	49.0	<0.001
Endurance	2.65 (1.22)	3.43 (0.99)	4.35 (0.63)	3.11 (1.24)	3.05 (0.76)	3.05 (0.83)	2.80 (0.95)	2.95 (0.97)	3.81 (0.66)	3.28 (1.05)	46.9	<0.001
Intelligence												
Pro-rated IQ	99.7 (11.2)	99.9 (13.5)	110.8 (13.6)	100.4 (12.8)	106.23 (14.5)	109.60 (11.6)	98.2 (12.7)	100.3 (10.9)	103.7 (14.0)	103.41 (13.4)	F=2.86	0.006

Table A4
Gender and IQ effects on questionnaire scores

	Gender			IQ
	Boys (N = 90)	Girls (N = 93)		
	Mean(SD)	*Mean(SD)*	*U* [§]	*r* [#]
Conners Teacher Rating Scale				
A. Oppositional	52.1(10.2)	52.7(11.8)	3358 *	-0.029
B. Cognitive Problems	51.2(8.9)	53.6(9.2)	3434 *	-0.240 **
C. Hyperactivity	50.3(8.6)	51.0(9.8)	3668	-0.031
D. Axious-Shy	57.1(9.9)	57.3(10.3)	4071	-0.015
E. Perfectionism	55.4(8.8)	53.6(7.2)	3680	0.131
F. Social Problems.	50.1(8.0)	52.6(10.2)	2909 **	-0.111
H. ADHD Index	50.1(8.1)	50.9(11.3)	4123	-0.163 *
I. GI: Restless-Impulsive	50.6(8.8)	50.9(11.3)	3989	-0.094
J. GI: Emotinal Lability	51.9(11.1)	51.7(10.2)	3539	-0.021
K. GI: Total	51.1(9.5)	51.4(11.1)	4067	-0.087
L. DSM: Inattentive.	50.4(8.6)	51.3(9.7)	3889	-0.206 **
M. DSM: Hyperactive/Impulsive	49.2(8.9)	50.5(10.3)	3381 *	-0.079
N. DSM: Total	49.9(7.8)	51.1(10.1)	4039	-0.196 **
Overall mean	51.5(6.3)	52.2(7.5)	4003	-0.136
Strength and Difficulties Questionnaire				
Emotional Symptoms	1.80(2.02)	2.15(2.48)	3890	-0.063
Conduct Problems	1.40(1.70)	0.95(1.80)	3202 **	-0.080
Hyperactivity	3.55(2.82)	2.23(2.66)	2875 ***	-0.131
Peer Problems	1.72(2.11)	1.45(2.12)	3625	-0.120
Prosocial Behaviour	6.72(2.34)	7.59(2.65)	2916 **	0.180 *
Total Problem score	8.52(5.89)	6.89(6.53)	3251 *	-0.162 *
Global ratings				
Ambition	3.43(1.03)	3.53(0.93)	4009	0.247 **
Endurance	3.19(1.03)	3.37(1.08)	3666	0.147 *

Note. [§] Mann-Whittney U-Test; [#] Spearman rank correlation
* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

7 Lebenslauf

Ausbildung

1963 – 1972	Primar- und Bezirksschule in Wettingen
1972 - 1977	Lehrerseminar in Wettingen
1979 - 1983	Musikstudium am Konservatorium Winterthur, Hauptfach Gitarre
1984 - 1987	Musik-Akademie Basel (Konzertklasse Gitarre)
1987 – 1996	Studium an der Universität Zürich
	Hauptfach Klinische Psychologie (Abteilung Schlaf- und Traumforschung), Nebenfächer Psychopathologie und Neuropsychologie. Abschluß mit Lizentiat.
1999 – 2002	Postgraduale Weiterbildung Psychotherapie mit kognitiv-behavioralem und interpersonalem Schwerpunkt (Universität Basel) mit Abschluss als Pychotherapeut FSP und Master of advanced Studies in Psychotherapy.

Berufliche Tätigkeiten, Praktika

1977 – 2000	Musiklehrer an der Kantonsschule Baden

Neuropsychologische und neurophysiologische Forschungstätigkeit

1994 – 1995	Mitarbeit im EEG-Brain-Mapping Labor der PUK Bern unter der Leitung von Prof. M. Koukkou-Lehmann.
1996 – 1997	Nachdiplompraktikum in klinischer Neuropsychologie am Universitätsspital Zürich, Neuropsychologische Abteilung der Neurologischen Klinik, Leitung PD. Dr. M. Regard.
1999 – 2002	Universitätsspital Basel, Neurologische Poliklinik: Neuorpsychologische Forschungstätigkeit über Rehabilitation von Aufmerksamkeitsstörungen bei Multipler Sclerose. Wissenschaftliche Mitarbeit (30%), ab Sept 2000 Leitung einer Multizenterstudie.
2003 – 2009	Forschungstätigkeit am Zentrum für Kinder- und Jugendpsychiatrie der Universität Zürich mit Schwerpunkt Neuropsychologische Grundlagen der Aufmerksamkeitsdefizit- und Hyperaktivitätsstörung ADHS.

Klinisch-psychologische Tätigkeit

1999 – 2000	Arbeit als klinischer Neuropsychologe am Universitätsspital Basel, Neurologische Klinik (Postgraduiertenstelle).
2000 – heute	Arbeit als Psychotherapeut