MOTOR PERFORMANCE AND MOTOR LEARNING IN ADULTS WITH DYSLEXIA

Jamie Luke Needle

Department of Psychology, University of Sheffield.

PhD Thesis

December 2005

Acknowledgements

I would very much like to thank Rod Nicolson and Angela Fawcett for their supervision of this work. I am very grateful for their help and support.

I would also like to thank DDAT, the sponsors of my PhD studentship, for their generous financial support.

Some of the experiments reported in this thesis are described elsewhere (Needle *et al.*, in press)

1. Abstract

Assessment of motor performance and motor learning in dyslexia is crucial because of its ability to shed light on the underlying biology of the disorder and to discriminate between theoretical approaches. It remains a controversial area due to existing discrepant research findings and interpretations.

Three studies are described in this thesis. The first used three sets of experiments to test balance and postural control in single and dual-task conditions. The second study examined the production and timing of responses in a classical eyeblink conditioning paradigm. The final study investigated motor skill acquisition. The results of the three studies were similar in that in dual-task balance, conditioned response timing and motor skill consolidation around half of the dyslexic adults showed substantial deficits compared with a control group.

The samples of participants in the three studies overlapped sufficiently for some cross-study comparisons of strengths and weaknesses to be conducted. These showed that it was rare for a participant with dyslexia to show motor impairment in just one of the three domains, with dual task balance and conditioned response timing seeming to be most closely associated.

Overall the results provide strong evidence of enduring deficits outside the literacy domain in dyslexia and also highlight the considerable heterogeneity of the disorder. Consequently they lend particular weight to the notion of cerebellar causation.

Further studies should be undertaken on a larger scale to scrutinize the consistency of motor impairments in dyslexia and the possibility that those showing motor problems might form a definite subgroup within dyslexia. In the longer term, this work points to a possibility of multiple, independently diagnosable sub-classes of dyslexia, based on specific neurological abnormalities, with their own specific remediation and objective early detection schemes.

2. Table of contents

3. A general introduction and review of the literature

3.1. Introduction

Dyslexia is a heavily researched and widespread disorder that can have severe consequences for the education and development of those who experience it, yet its cause remains unclear, possibly because of the tendency to focus research on literacy to the neglect of other facets of dyslexia. The three studies reported here are framed within the major theories of dyslexia (phonological or double deficit, magnocellular deficit and cerebellar deficit), and investigated motor performance and/or motor learning in dyslexic adults. There was considerable overlap in the participant samples employed by the three studies. The general aim was to examine performance of adults with dyslexia on the various tasks compared with age and IQ matched control participants. Secondary to this were a further two general aims, to establish the prevalence of impairments in these tasks, and to record the consistency of deficits across studies for each participant.

The following review of the literature will begin by addressing dyslexia definition, which will lead inevitably to the various existing explanatory theories. These will be described and considered in some depth, before a shift in emphasis to some critical gaps in knowledge with respect to dyslexia theory, that are the focus of this thesis. Briefly, these are 1) balance, posture and automaticity; 2) the cerebellum and classical eyeblink conditioning; 3) learning and consolidation; 4) subgroups. Potential implications of investigations in these areas will be assessed for each of the explanatory frameworks described.

A research area so large and broad as dyslexia research will be prone to causing confusion. To guard against this it is important to set out some 'ground rules' in terms of the different levels of explanation (Frith, 1997), which should be acknowledged and employed explicitly. The principal levels are the *biological*, *cognitive* and *behavioural* levels. While it is often appropriate and convenient to discuss a disorder like dyslexia at one level at a time, a one-level account should not be thought of as much more than a description. Explanation comes as causal links are made between the levels, and these links (just as the within-level descriptions) should be backed up with empirical observation. Furthermore, it should be borne in mind that a certain description at one level might be caused by any number of possibilities at the next level. Finally, it may also be appropriate to employ further levels of explanation, for example a *genetic* level of explanation beyond the biological level (Nicolson, 2001) or a *neural systems* level between the cognitive and biological levels (Nicolson & Fawcett, Submitted). Genetics is a rapidly progressing area of dyslexia research, but here too the emerging picture is not clear-cut, for example, Fisher and Smith (2001) comment that "It is very likely that dyslexia is genetically heterogeneous, meaning that there are different genes (possibly with different transmission patterns) predisposing to reading disability in different families." (p. 42).

Dyslexia has received vast research attention not least because of its very high prevalence. Estimates for English speaking countries range from 2% to over 10% depending on the criteria used (Badian, 1984; Jorm *et al.*, 1986; S. E. Shaywitz, 1998). Due to the lack of understanding and consensus at other levels, dyslexia is defined at the behavioural level which gives rise to the possibility of widely varying diagnostic criteria. For this thesis I have adopted the following definition from the World Federation of Neurology: "a disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities" (World Federation of Neurology, 1968). The definition was operationalized as described in section 4.2.1.2. However, even this definition would be disputed by some, since it assumes a discrepancy between literacy attainment and performance in other cognitive domains. Some would argue that persons with difficulty learning to read should not be separated on the grounds of the existence or otherwise of this IQ discrepancy (for a discussion of dyslexia definition including this point see Lyon *et al.*, 2003). This argument is usually based on the apparent similarity between poor readers with and without IQ discrepancy in terms of a cognitive level assessment of their failed learning to read, specifically the common element of a lack of phonological awareness. However, Demonet *et al.* (2004) have pointed out that the debate on the use of a discrepancy criterion should be informed by the fact that there seems to be a stronger genetic link with reading disability where the individuals do have relatively high IQ than in the cases of non-discrepant poor readers. On the other hand, van Daal and van der Leij (1999) have argued that the important discrepancy is with listening comprehension or verbal competence rather than IQ. Whatever the result of the IQ discrepancy debate, the phonological deficit of poor readers is certainly a key element of dyslexia, and is at the heart of what has been the dominant theory in the field for the last 20 years, and to which I now turn.

3.2. The phonological deficit hypothesis

A prerequisite for learning how to read or spell is the ability to break words down into their constituent parts. In turn this requires an ability to perceive the separable sections of a word, or put another way to be able to distinguish the phonemes within a spoken utterance. The Phonological Deficit Hypothesis (PDH) of dyslexia sprang from the observation (e.g. Bradley & Bryant, 1983; Snowling, 1981) that children with dyslexia lacked this sensitivity to phonemes. There has since been consistent support for the lack of phonemic awareness in individuals with dyslexia (e.g. Nicolson & Fawcett, 1995; Snowling, 1995), even into adulthood (Ramus *et al.*, 2003b). In fact, the latter researchers found phonological deficit extremely pervasive in their sample of dyslexic students, and asserted that phonological deficit is always sufficient to cause dyslexia (even where there are no other low-level 'symptoms'). Perhaps more important than poor phonological awareness producing reading problems, programs designed to improve phonological awareness have achieved gains in reading and spelling (e.g. Eden *et al.*, 2004; Torgesen *et al.*, 1999). The phonological perspective acknowledges that some dyslexic children may well

exhibit other difficulties but has the strong belief that the phonological domain reflects the 'core' disability (Catts, 1996; S. Shaywitz, 1996; Stanovich, 1988).

The phonological framework then, has been extremely fruitful in that it has described an underlying cognitive phenomenon (phonological awareness) that appears to be so strongly predictive of dyslexia that addressing it also addresses the key practical impairments, those of reading and spelling. However, the ending of the key symptoms in a dyslexic individual does not mean that the person in question is "cured". This slightly counter-intuitive statement reveals a major limitation of this phonological framework, the phonological and literacy problems experienced by people with dyslexia are not the sum of the disorder. Even in a fairly conservative study of adults with dyslexia, Ramus and colleagues found a striking heterogeneity of abilities and impairments (Ramus *et al.*, 2003b). On the biological level there must be some difference between people who are dyslexic and people who are not, this is almost guaranteed by the definition adopted above, which rules out lack of education as a cause. Furthermore, the phonological deficit hypothesis as described above does not account for the dyslexic's characteristic processing speed (Fawcett & Nicolson, 1994) and working memory (McLoughlin *et al.*, 1994) deficits, or indeed other well-documented impairments further removed from literacy (Fischer *et al.*, 2000; Moe-Nilssen *et al.*, 2003; Stoodley *et al.*, 2000). On this point, Nicolson and Fawcett (Submitted) have asserted that verbal working memory and processing speed are "fundamental cognitive attributes" that cannot satisfactorily be assumed to be part of a phonological awareness problem, but rather are independent of phonology. So while it is a helpful tool for the investigation of elements of dyslexia most closely related to reading and spelling, the pure phonological deficit hypothesis fails to encompass elements of dyslexia (working memory and processing speed deficits) that even the most conservative theorists would consider fairly fundamental to the disorder. On the other hand, phonological deficit is not specific to dyslexia, but also characteristic of non-dyslexic poor readers as alluded to above in the consideration of dyslexia definition, (Siegel, 1989; Stanovich & Siegel, 1994).

3.2.1. Double deficit

This problem doubtless led Wolf and Bowers (1999) to the formulation of their *Double Deficit* hypothesis, which asserts that processing speed (alongside phonology) is a separate central area of dysfunction in dyslexia. It is possible to show impairment in either area alone, with the most severe dyslexia being exhibited where there are both phonological and speed deficits. There is face validity to this theory in that reading fluency failure is just as significant a feature in dyslexia as are reading inaccuracies. Indeed, dyslexic persons in countries where the language's orthography is more regular show far fewer phonological problems than do dyslexic English speakers, leaving speed as the major issue. The classic measure of speed in this context is the Rapid Automatized Naming task, which was first used to test people with dyslexia by Denckla and Rudel (Denckla & Rudel, 1976) who reported a speed deficit in dyslexic children. Processing speed deficits have since been shown in other tests (Nicolson & Fawcett, 1994b; Yap & van der Leij, 1993) and have

become part of some diagnostic testing batteries (e.g. Nicolson & Fawcett, 1996, 1997).

3.2.2. Biological causation

A clear biological level explanation of dyslexia is important for pure theoretical completeness, but would also have weighty applied significance. Firstly, because a more complete understanding of the disorder might enable more fundamental approaches to remediation. It is conceivable that at least some persons who are now diagnosed as dyslexic might be helped by some biologically based intervention. Secondly, a biological cause may well have a marker that is clearly detectable well before school age, perhaps even at birth. Were this found to be the case, it would be possible to know which children to expect to struggle with reading before they even began to try, and therefore suitable support could be in place before they fell behind.

It is fair to say that the vast majority of research within the PDH framework has been concerned with the cognitive level to the detriment of the biological level. However, traditional phonological deficit theorists suggest that the difficulties originate within language areas of the cerebral cortex or perhaps as a disconnection between cortical regions (Eden *et al.*, 2004; Rumsey *et al.*, 1999; B. A. Shaywitz *et al.*, 2002). Specifically, Galaburda and colleagues (1989) investigated the brains of deceased persons who were known to have been dyslexic and reported ectopias and scarring in the perisylvian area. Until more recently there was no clear description of how these were related to the complex behavioural symptoms of dyslexia, however Ramus (2004) has recently provided some insight to this end, drawing on some intriguing findings and ideas described by Galaburda himself (Galaburda, 1999).

3.3. Sensory processing deficit

Over the past 30 years a considerable body of evidence has emerged indicating auditory problems in people with dyslexia. A significant pre-cursor to this work was the finding that children with Specific Language Impairment (SLI) perform poorly on tasks of temporal order judgment (Tallal & Piercy, 1973). Tallal and colleagues proposed that a similar deficit in sensory processing speed could lead to phonological awareness difficulties in dyslexia (Tallal *et al.*, 1993). Functional MRI has revealed unusual activation patterns in persons with dyslexia who were undertaking a task involving processing of rapid acoustic stimuli (Temple *et al.*, 2000). Decreased sensitivity to auditory frequency modulation is not confined to English speaking poor readers but also applies where orthography is more regular (Talcott *et al.*, 2003). Recently Hulslander *et al.* have published findings somewhat supportive of the notion of auditory processing abnormality in dyslexia, but they raise the need for caution in undertaking such studies, noting that most tests of sensory processing are not independent of IQ or attention (Hulslander *et al.*, 2004).

Visual problems have also been reported in dyslexia, in terms of saccadic control as well as sensory processing (Biscaldi *et al.*, 2000; Biscaldi *et al.*, 1998; Fischer et al.,

2000). Key early discoveries were made by Lovegrove and colleagues (Lovegrove *et al.*, 1980; Martin & Lovegrove, 1987) who observed that dyslexics had lowered sensitivity to contrast and flicker. A particularly strong finding in this area of visual perception was Talcott *et al.*'s (1998) report that dyslexic deficit in sensitivity to motion and flicker was both strong and universal enough to discriminate 73% of the dyslexic participants in the study from the control subjects. These researchers have gone on to publish further experiments suggesting a strong relationship between motion sensitivity and measures of literacy (Talcott *et al.*, 2003; Talcott *et al.*, 2000; Witton *et al.*, 1998). Much work on the motion sensitivity strand of the visual processing deficit in dyslexia was carried out by an Oxford research group in the early nineties (e.g. Cornelissen *et al.*, 1995).

It is clear that impaired input in either the visual or auditory modalities could plausibly produce difficulties in acquisition of reading and writing skills. In particular, 'fuzzy' timing of auditory input perception could make it very difficult for someone to attain normal phonological awareness. However, as described above, there is much more to dyslexia than the criterial literacy areas. In answer to this point, some researchers (both Stein and Tallal for example) have proposed that dyslexia is caused by a cross-modal sensory processing deficit. This proposition is supported by the finding of correlation of auditory and visual impairments (Witton *et al.*, 1998) and the case is aided further by a study of vibrotactile sensitivity (Stoodley *et al.*, 2000) that reported dyslexic deficit at a specific low frequency of vibration but not at higher frequencies, indicating sensory abnormality in a further modality.

3.3.1. Biological causation

Furthermore, Stein (Stein, 2001; Stein & Walsh, 1997), has proposed that a general sensory processing deficit like that alluded to above, is attributable to deficient magnocellular processing. The visual system is made up of two distinct subsections, the magnocellular system deals with information on motion while detail and colour are dealt with by parvo cells. Stein and Walsh (1997) reasoned that since the magnocellular stream runs to the parietal cortex, the magnocellular deficit hypothesis (MDH) of dyslexia is supported by various categories of experiment that have implied an impairment of visual attentional systems in dyslexia. Although it is less clear and less well described, a similar magno-parvo type distinction is thought to exist for audition and possibly for the other senses. So if there were some general abnormality of the magnocellular system, one could expect there to be impairments of sensitivity to motion and timing in all senses, similar to those mentioned above. In turn these would hamper acquisition of phonological skills (through formation of weak or inaccurate representations). Furthermore, such a fundamental rapid processing deficit could also produce the classic processing speed problems, whereas noisy auditory input would increase the demands on verbal working memory for any given task compared with the demands experienced by someone with normal magnocellular function. If the pan-sensory impairment extends to tactile stimuli, then this coupled with the established visual motion problems could be expected to generate the clumsiness that practitioners have often described in

children with dyslexia. In short, the general sensory processing deficit notion accounts for a broader range of dyslexic symptoms than can the phonological approach.

Empirically there is some consistent evidence, for example, further investigation of the dyslexic brain bank that Galaburda studied (see section 3.2.2 above) revealed that dyslexic brains contained fewer and smaller magno cells in the lateral geniculate nucleus. No corresponding difference was seen for parvo cells (Livingstone *et al.*, 1993). In an fMRI study, Eden *et al.* (1996) observed differences between dyslexic men and controls in the activation of portions of the visual magnocellular system in response to moving stimuli. Digging deeper confronts the question of why there is a magnocellular abnormality in these people. The rapid response of magno cells that is a necessary adaptation for their function in timing, is aided by a high local concentration of flexible unsaturated fatty acid chains (Stein *et al.*, 2001). High levels of the enzyme which removes these acids have been observed in dyslexics (MacDonnell *et al.*, 2000). On these grounds, researchers are investigating the possible efficacy of polyunsaturated fatty acid dietary supplements for the facilitation of literacy skill acquisition.

3.3.2. Limitations of the MDH framework

There are two major challenges for this framework that should be discussed at this point; *behavioural prevalence* and *alternative biology.*

(i) *Behavioural prevalence.* As described above, several studies have reported specific deficits in visual or auditory processing in people with dyslexia, but on an individual by individual basis, these seem to be the exception rather than the rule. Ramus (2003) estimates rates of visual and auditory deficit in dyslexia of 29% and 39% respectively (this is consistent with the 30%-40% rate of occulomotor abnormalities in dyslexia discussed by Biscaldi *et al.* (2000)). So this is not an insignificant phenomenon, but if as the theory seems to suggest, the hypothesized impairments in the input systems lead **directly** to phonological impairments, it is puzzling as to why the remaining 61% to 71% of participants in these studies show dyslexic symptoms without sensory input problems. In addition, it would be reasonable to expect those showing sensory deficits in one modality to show deficits in at least one other. This has not been consistently reported to the author's knowledge, but certainly deserves further experimental investigation (Witton *et al.*, 1998).

(ii) *Alternative biology.* Some of the phenomena that proponents of the MDH would attribute to poor magnocellular sensory input are perhaps more naturally explained another way, particularly with reference to the cerebellum, whose considerable role in sensory input integration is only recently being acknowledged (Bower & Parsons, 2003). In other words, it may be that the problem is not so much to do with the input pathways but with their target (or both). The cerebellum is certainly a significant target of magnocellular information (Stein & Walsh, 1997). Indeed, in a wide-ranging exposition of his theory, Stein refers to the cerebellum as the "head-ganglion" of the magnocellular system (Stein, 2001, p.13). The necessary

discussion of the cerebellum in a complete magnocellular theory of dyslexia is further illustrated by Moretti *et al.* (2002) who note that the reading errors of patients with cerebellar vermis lesions could be because of the structure's interaction with frontal lobe language areas or simply because of its involvement in ocular control, of course, either type of dysfunction could be related to visual/magnocellular problems.

3.4. Automaticity deficit

So at the cognitive level there is considerable evidence for a crucial phonological deficit, which may be the product of a wider cognitive level construct, that of general sensory processing deficit. However, this conceptualization appears to presume that acquisition of phonological awareness (and then reading) from normal sensory processing is a natural progression, neglecting the fact that reading is a complex learned skill. Indeed, phonological awareness itself can be taught, and this is a major positive outcome of the PDH framework. Moreover, reading is a skill that must be learned to the point of considerable fluency and automaticity: "Thus, in order to comprehend what one reads, one must be able to identify the words contained in running text with enough accuracy and fluency to allow computation of the meanings embodied in the text within the limits of working memory." (Vellutino *et al.*, 2004, p. 5).

This vision of reading as a skill was a catalyst for Nicolson and Fawcett's (1990) early work on balance in dyslexia, which produced the Dyslexia Automatization Deficit hypothesis (DAD). In those experiments the researchers tested the balance skill of dyslexic adolescents and controls in various conditions, some of which included the requirement to simultaneously perform a secondary cognitive distracter task (e.g. counting). The major finding was that the participants with dyslexia showed marked balance impairment when performing a secondary task but not in balance-only conditions. The authors interpreted this as evidence for a failure on the part of the participants with dyslexia to have fully automatized balance, with the poor performance in dual-task conditions a consequence of the prevention of conscious compensation for the lack of balance automaticity. They further speculated that this might be indicative of a general failure to automatize skills, including reading. The idea of automaticity failure fits nicely with the classic impairment of Rapid Automatized Naming reported by Denckla & Rudel (1976). The effect of an automatization deficit in skill acquisition should be expected to be cumulative as the target skills become more complex and are made up of more and more component sub-skills. A standard mark of automatic performance is the ability to perform two tasks simultaneously without interference between the two, so that they are performed as if they were being undertaken one at a time. Automatic performance therefore facilitates the blending of basic sub skills into a more complex behaviour. Nicolson and Fawcett (2000) have reported a deficiency in this procedure in people with dyslexia at a very low level, that of merging two simple reaction tests into a two-choice reaction test. More recently, there has also been a report of a failure to attain automatic shape recognition in dyslexia (Moores *et al.*,

2003). A weakness of automaticity as an explanatory term is that it is rather vague – there is a consensus over its general meaning, specifically automatic performance must be resistant to interference for example, but since it is a term applied to a great diversity of tasks there will inevitably be confusion and questions as to its precise meaning. Additionally, automaticity seems to be quite a broad term. This is reflected in Nicolson and Fawcett's (2000) report of normal 'strength' of automatization in a dyslexic group, but impaired 'quality' of automatic performance.

3.4.1. The cerebellum and dyslexia

In terms of the biological level of explanation, the existence of an automaticity deficit in dyslexia is consistent with a cerebellar connection. In his review of language and the cerebellum, Fabbro (2000) notes that the cerebellum is "...responsible for learning and automatizing complex motor sequences." (p. 90). More on the topic of the cerebellum's role in motor automaticity follows throughout this thesis. Here the structure will be introduced more generally.

The cerebellum is a dense but highly uniform subcortical brain structure, containing around half of all the brain's neurons. The cerebellar cortex is divided into left and right hemispheres and, like the cerebral cortex, is very much enlarged in humans compared to our evolutionary predecessors. Output from the cerebellar cortex goes via the cerebellar deep nuclei (dentate, interposed and fastigial), according to the originating area of cortex (lateral hemisphere, medial hemisphere and vermis respectively) (Makris *et al.*, 2003). Traditionally the cerebellum was supposed to be involved purely in motor functions, particularly in the coordination and smoothening of movements and achievement of automaticity (e.g. Lang & Bastian, 2002). In the light of a large body of research evidence there is now widespread acceptance for the idea that the cerebellum has a much broader range of influence and that it is involved in many cognitive processes (Desmond & Fiez, 1998; Fabbro, 2000; Leiner *et al.*, 1993; Rapoport *et al.*, 2000; Riva, 2000; Silveri & Misciagna, 2000; Thach, 1998), including reading (Fulbright *et al.*, 1999; Moretti *et al.*, 2002; Seki *et al.*, 2004). Given the uniformity of structure in the cerebellar cortex, one might speculate that the cerebellum performs a similar style of operation on the diverse inputs it receives, achieving coordination and fluency of performance. The anatomical architecture clearly facilitates negative feedback or "supervised" learning (cf supervised learning in Doya (2000)) as noted by early theorists (Albus, 1971; Marr, 1969), and proposed for eyeblink conditioning. Here the conditioned stimulus is thought to be conveyed to interpositus nucleus and cerebellar cortical Purkinje cells via mossy fibres. At both locations it converges with information about the unconditioned stimulus arriving from the inferior olive by way of climbing fibres, whose complex spike signals exert a profound effect on Purkinje cell dendritic trees. Comprehensive accounts are provided by the following publications (Gluck *et al.*, 2001; Hesslow & Yeo, 2002; Steinmetz, 2000). Importantly, error-related signals have been recorded in cerebellar cortex in an fMRI study of human eyeblink conditioning (Ramnani *et al.*, 2000).

Researchers in Sheffield have undertaken a broad range of experiments to test the hypothesis that abnormal cerebellar function underlies dyslexia (the Cerebellar Deficit Hypothesis or CDH, Nicolson *et al.*, (2001)). These have generated a significant body of general support and include classic clinical cerebellar tests (Brookes & Stirling, 2005; Fawcett & Nicolson, 1999; Fawcett *et al.*, 2001b), time estimation (Nicolson *et al.*, 1995), classical eyeblink conditioning (Nicolson *et al.*, 2002), motor sequence learning with neuroimaging (Nicolson *et al.*, 1999) and visual adaptation (Rebecca Brookes, personal communication). Furthermore, neuroscientists have discovered anatomical (Rae *et al.*, 2002; Eckert *et al.*, 2003; Eckert *et al.*, 2005) and biochemical (Rae *et al.*, 1998) differences between the cerebella of dyslexics and controls. However, questions have been raised over the interpretation of some of this data (Beaton, 2002). Of course there is considerable overlap between 'cerebellar' and 'magnocellular'/'visual'/'auditory' tasks. The cerebellum is centrally involved in saccade generation and the vestibular-occular reflex, indeed Makris *et al.* (2003) mention regulation of eye movements as one activity particularly related to cerebellar vermis/flocculus. Consequently a cerebellar explanation might fit some of the data alluded to under earlier sections and vice-versa. For example, one area of the unusual pattern of activation seen in dyslexics during an acoustic rapid processing task discussed above (Temple *et al.*, 2000) was the right posterior cerebellum.

3.4.2. Causal chain

Fawcett and Nicolson (Fawcett & Nicolson, 2001; Nicolson *et al.*, 2001) have proposed a comprehensive theory outlining how impaired function of the cerebellum and/or of a cortico-cerebellar loop could produce the considerable range of behavioural symptoms of dyslexia. Balance and motor skill impairments are accounted for naturally, since the cerebellum has long been known to be part of the vestibular system and integrally involved in motor coordination. However in their "ontogenetic causal chain" (Nicolson *et al.*, 2001), these researchers further propose that difficulties in the complex motor skill of articulation lead to impaired phonological awareness while the cerebellar impairment itself causes general problems in automatizing skill and knowledge. In the model, these automatization deficits combined with the phonological deficit derived from articulation difficulties produce impairments in grapheme-phoneme conversion, the word recognition module, verbal working memory and learning of orthographic regularities. These in turn, inevitably result in poor reading and spelling.

3.4.3. Criticisms of the CDH framework

The CDH framework has gained a measure of acceptance since much early controversy, but is still not taken to be authoritative by the majority. Some reasons for this are discussed below:

(i) *If cerebellar impairment produces skill deficits across the board, why is the reading deficit so prominent while others are often elusive?* There are a few possible answers to this point.

a. Firstly and briefly, it should be noted that reading is one of the most tested skills that a child learns, and is also considered one of the most important. Consequently difficulties in acquiring this skill are most likely to be noticed and taken seriously. As an illustration, note that a school child is not given additional training if s/he is seen to have football ability typical of someone 2 years younger. So other skill deficits may often go unnoticed.
b. Secondly, the issue of cumulative deficit in complex skills is important. Reading is a complex skill, which any child learns through a progression of stages, with each level needing to be mastered (ideally automatized) before the next stage is embarked upon. If none of the sub-stages are fully automatized cumulatively increasing deficits should be expected with the beginning of each new stage. According to Fawcett and Nicolson's (2001) ontogenetic causal chain, articulation is one prerequisite in normal reading development. In their review, Ackermann and Hertrich (2000) outline the cerebellum's contribution to speech processing, but also make the point that verbal communication can demand the control of more than one hundred muscles. This is no small feat. Moreover, Fawcett and Nicolson (2002) observed slowed articulation in a group of dyslexic children.
c. Thirdly, researchers are good at testing reading. Often less well researched areas of skill are not examined so successfully through a relative lack of practice and expertise. Floor and ceiling effects are particular possibilities. This point is returned to later (e.g. section 3.7.2.).

(ii) *The cerebellar deficit hypothesis lacks specificity, this is such a large and under-researched structure that a cerebellar deficit hypothesis is not very informative.* This question will be addressed further throughout the thesis, at this point it will suffice to say that the cerebellum's wide-range of influence may be a particular theoretical advantage because the full behavioural manifestations of dyslexia are so diverse and inconsistent from one individual to the next. Perhaps varied but overlapping cerebellar deficits are the causes of various subtypes of dyslexia. Much more research is clearly needed on this point.

(iii) *The theoretical link from articulation to phonological awareness is not empirically supported.* This point is made by Ramus and colleagues (Ramus *et al.*, 2003a, p.720), on the basis of the case of a patient with severe congenital dysarthria who showed no problems in the acquisition of literacy. However, it is unlikely that the normal process of literacy acquisition is modelled accurately in this patient's case. Although normal articulation may not be necessary for the development of phonological awareness it is clear that in a typical case the two develop in synergy.

(iv) *CDH evidence is inconsistent, e.g. Ramus et al.'s (2003a) study of dyslexic children.* In a disorder so widespread and behaviourally diverse as dyslexia it is unwise to assume that all cases share a single biological

cause, but it should be noted that cerebellar dysfunction could cause phonological difficulties (via slowed articulatory development) whether or not motor symptoms were detectable. Furthermore, studies investigating 'cerebellar' signs in people with dyslexia have consistently found impairments in at least a substantial minority of dyslexic participants.

3.5. Co-morbidity

It has been a theme above that the manifestations of dyslexia are not restricted to literacy, but exist in an extremely wide array of domains. Many of the characteristics of dyslexia that have been observed, e.g. motor coordination (Fawcett & Nicolson, 1995; Moe-Nilssen et al., 2003) or attentional abnormalities (e.g. Facoetti *et al.*, (2001)) are key characteristics of other supposedly discrete conditions, e.g. developmental coordination disorder (DCD or dyspraxia) and attention deficit hyperactivity disorder (AD(H)D) respectively. There is, then, considerable behavioural overlap between these conditions. Add to that the fact that these disorders are behaviourally defined, and the borders between them suddenly appear rather weak and perhaps somewhat arbitrary. Kaplan *et al.* (Kaplan *et al.*, 2001) have published a helpful article on this matter, insightfully entitled "The term co-morbidity is of questionable value in reference to developmental disorders: data and theory". Here they present evidence of very high co-morbidity rates, for example, of their sample of 126 children meeting criteria for reading deficit (dyslexia), 63 were also diagnosable on criteria for ADHD, and 20 met the criteria for DCD. This is an even greater overlap than reported by Wimmer *et al.* (1999) who assigned 10 of their 30 dyslexic participants (and 6 of 30 controls) to an ADHD subgroup. This proportion (33%) is still higher, however, than the 15% suggested in the mid nineteen-nineties (S. E. Shaywitz *et al.*, 1994).

This high co-morbidity must have implications for theorising, yet it is hard to see how a purely phonological dyslexia, caused by abnormality of cerebral language areas, could account for the range of dyslexic symptoms, let alone all the symptoms of these apparently related disorders. It remains a possibility that some minority of dyslexic people have purely cerebral cortical, phonological deficit dyslexia, but it seems that the majority of those suffering reading difficulty despite normal education and intelligence are endowed with a much more complex set of strengths and weaknesses. Turning to the two theories that invoke further brain regions in detail, the CDH framework would predict the observed range of difficulties (from balance to articulation to digit span to skill blending to classical conditioning to reading) since the cerebellum is hypothesized to be involved to some extent in all the relevant areas of processing, particularly those traditionally in the DCD arena. The MDH framework could also accommodate them, but would be hard pushed to do so without reference to a dysfunctional cerebellum as the system's "head-ganglion" (Stein, 2001, p. 13). Why would fuzzy sensory input alone produce impairment in skill blending for example?

The idea that dyslexia, dyspraxia and attention deficit disorder are all caused by sub-optimal cerebellar development is the driving philosophy for the DDAT centres, which offer an exercise based remediation program commercially. Published research (Reynolds *et al.*, 2003) has corroborated the organisation's claims as to the treatment's efficacy, but it was met with much hostility in the academic community (Rack, 2003; Snowling & Hulme, 2003).

Regardless of the cause(s) of these disorders, few would dispute that they commonly co-occur, which raises the possibilities that their causes are related or that they exist on a continuum with a common set of interacting causes. Consequently it is important not to study them in isolation but to examine how they inter-relate behaviourally to further investigate these possibilities. Related ideas are considered in a later section on subgroups (section 3.10.).

3.6. Conclusions from the background literature

The phonological deficit framework has proved fruitful in describing the cognitive precursors to reading failure and consequently, in inspiring successful remediation methods for those already suffering particular reading difficulties. However it falls short of offering a complete and coherent explanation of dyslexia. Two broad frameworks (centred around pan-sensory magnocellular deficit and cerebellar deficit) have attempted to do this. Both incorporate impaired phonological awareness as a crucial cognitive level determinant of reading difficulty and both have produced a range of supporting evidence. The cerebellar deficit theory seems to have the advantage in terms of ability to naturally account for the diversity of symptoms and also in terms of prevalence of signs of cerebellar deficit. On the other hand it is perhaps not specific enough, leaving itself open to criticisms of weak predictive power or unfalsifiability.

Consideration of the state of research aimed at refining and comparing these theories of the cause(s) of dyslexia reveals some key gaps in current scientific knowledge. Addressing these is the focus of this thesis. Each will now be introduced and discussed in turn together with the relevant literature, the reasons for their importance and the implications their investigation may raise for the major theoretical frameworks.

3.7. Issue 1: Balance, posture and automaticity

Balance has great theoretical significance in dyslexia research, for two principle reasons.

(i) The theory that dyslexia is caused by a general failure to automatize skills has its foundations in the early Nicolson and Fawcett (1990) balance experiments. A general automatization deficit would extend to motor domains and therefore the observed dyslexic impairment in a skill so seemingly far removed from literacy as balance appears strong evidence for the existence of a general automaticity deficit. It is certainly

hard to explain from the perspectives of phonological or sensory processing causations.

(ii) Balance deficit can also be taken as indicative of cerebellar dysfunction, both directly and indirectly. As outlined throughout this chapter, the cerebellum seems to be involved in the learning of a diverse range of skills, particularly where automaticity is approached. Therefore an established motor automaticity impairment could point to cerebellar dysfunction. From a direct perspective, it should be noted that the cerebellum is directly involved in the maintenance of tandem/heel-to-toe balance, as testified by both lesion and behavioural PET studies (Bastian *et al.*, 1998; Ouchi *et al.*, 1999).

3.7.1. Existing studies of balance and dyslexia

There have been surprisingly few attempts at replication of the foundational balance experiments. Yap and van der Leij (1994) using Dutch dyslexic children slightly younger than those in the former study (aged 10 rather than 13) provided a close replication. Participants' balance was rated while they stood on one leg and then again while the same balance task was performed simultaneously with an auditory choice task. Each test lasted 1 minute. The added demand of the secondary task produced decreased stability in the dyslexic group but not in either reading age or chronological age controls when right and left leg scores were combined in an analysis of variance. The authors concluded that their results "...replicate the findings of Nicolson and Fawcett (1990) and support the automatization deficit hypothesis." (p. 663). In contrast, a later study from the same research group found no support for the automaticity deficit position in the results of their motor and balance tasks (van Daal & van der Leij, 1999), but here the sampling criteria were relatively broad.

At a similar time, Wimmer and co-workers (1999) tested Austrian children (aged 8-10 years) using balance tests based on Nicolson and Fawcett (1990). Interpretation of these is clouded by differences in procedure, particularly regarding the specific posture adopted for the balance tests. In addition, the later study included teacher assessment of ADHD symptoms in the analysis of balance as well as dyslexia classification. This produced the finding that dual-task balancing was more difficult only for the dyslexic children with high ADHD scores, not for dyslexic children with lower ADHD scores. Apart from the procedural change noted above there is one considerable reason for caution in the comparison of the two studies, it is one of sampling. There was no official dyslexia assessment procedure in Austrian schools and children's classification in this study was based on lowered reading speed together with normal non-verbal IQ. So the discrimination was based on fluency without accounting for accuracy, probably because the German language is much more regular than English, but this raises the real possibility that the children referred to as dyslexic in the two studies were in fact not suffering the same condition.

Recently, Raberger and Wimmer (2003) have published further research on 10 year old Austrian children, which reaches a similar conclusion to that of their earlier work: "...poor balancing was associated with ADHD and not with reading disability, whereas poor rapid naming was associated with reading disability and not with ADHD." (Raberger & Wimmer, 2003, p. 1496,). Unlike the Fawcett and Nicolson (1992) study[1] and all their subsequent studies, the Nicolson and Fawcett (1990) study did not screen for ADHD.

A further study providing related findings, was conducted by Moe-Nilssen *et al.* (2003). These researchers studied 10-12 year old Norwegian children. They administered many measures of postural control to dyslexic and control participants, some of the tests examined standing balance/sway whereas others were concerned with motor control during walking. Of the 6 standing tests, 4 exposed dyslexic deficits in balance with the exceptions being the one where participants' eyes were closed and another where movement was externally provoked. The four unperturbed standing tests with eyes open correctly classified at least 70% of the subjects as dyslexics or controls. Walking parameters also revealed differences between the groups and could be used to correctly classify over 77% of the subjects.

Most recently, Stoodley *et al.* (2005) have reported balance difficulties in at least 50% of a sample of dyslexic children (aged 10) together with strong relationships between balance and reading and spelling performances. This study measured balance using Polhemus equipment employed in experiments described in this thesis (section 4.5.).

Poblano and co-workers (2002) used posturographic recording equipment similar to that used in study 1 experiment 1 (section 4.3.) to assess gross motor control in 9 year old children with learning disabilities in reading and writing using classification criteria from the DSM-IV. Children with learning disabilities showed some abnormalities of movement coordination but performed as well as controls in a sensory organisation test.

A final study with children was undertaken by Ramus, Pidgeon and Frith (Ramus *et al.*, 2003a). The researchers found that 50% of their dyslexic children showed problems in balance using the postural stability subtest of the Dyslexia Screening Test (Fawcett & Nicolson, 1996). These children were aged between 8 and 12.

3.7.2. Balance and dyslexia in adulthood

Clearly balance control mechanisms should be modified and adapted throughout development, since the subtleties of the task will change with changes in an individual's physical dimensions. Furthermore, the rapid growth associated with puberty occurs at varying times across individuals and could be expected to cause particularly strong demands on control mechanism adaptation. Therefore there can

[1] Which replicated the pattern of findings reported in (Nicolson & Fawcett, 1990).

be difficulties in interpretation of balance data in children. To some extent these difficulties are avoided in testing adults. It is assumed that by adulthood the task of postural control is relatively stable and all major developmental changes are complete or nearly complete. However, balance testing in adults brings its own difficulties, since balance control is generally well developed by adulthood it is important to use carefully chosen tests of appropriate sensitivity. Two footed balance (side-by-side) is generally not sensitive enough (generating ceiling effects) and one footed balance may be too difficult (leading to floor effects). For this reason the heel-to-toe or tandem stance is often adopted. Balance research in adults runs the risk of missing deficits in individuals who previously showed impairment but who have caught up by adulthood, but provides a usefully stringent test, in that any enduring balance deficits at adulthood can be taken to be long-term characteristics of dyslexia rather than mere delayed development.

A fully controlled study of dyslexic adults was undertaken by Ramus and colleagues (Ramus *et al.*, 2003b) who tested dyslexic university students and controls on a wide range of measures designed to investigate the generality of deficits predicted by the various theories of dyslexia. With reference to the Cerebellar Deficit Hypothesis they tested balance in four conditions: "(i) eyes open, feet apart; (ii) eyes closed, feet together; (iii) eyes closed, feet together, arms extended; (iv) eyes closed, feet together, arms extended and counting backwards" (p. 850). No difference was found between the dyslexic and control groups on these measures. However, as outlined above, interpretation of such null effects is clouded by the procedure. For instance, the "feet together" posture maintained by Ramus's student participants involved standing on balance monitoring equipment based on a standard bathroom scale pattern. That is, feet side-by-side rather than one in front of the other. This is a much easier task than heel-to-toe balance as it is sensitive only to front-to-back sway rather than side-to-side sway. The side-by-side stance also provides a larger more stable base. And consequently less scope for destabilisation. It is therefore likely that all participants were subject to ceiling effects. Moreover, it was impossible to investigate the central issue of interest (the question of whether balance was impaired in a dyslexic group when conscious compensation was prevented by a dual-task paradigm) because the condition where there was a counting task also had participants blindfolded. The blindfolding would be expected to increase variability in both groups and thereby hide any between group differences. Nonetheless, a particularly relevant issue highlighted by this research is that of 'co-morbidity', or heterogeneity: Four of the 16 dyslexics tested showed a motor deficit even in these conditions.

The study of balance reported in this thesis aimed to add clarity to the important and controversial issue of balance and postural control in dyslexia by testing adults through a variety of procedures, including the original paradigm employed by Nicolson and Fawcett (1990).

3.8. Issue 2: The cerebellum and classical eyeblink conditioning

Dyslexia is traditionally defined at the behavioural level (for example, the definition adopted for this thesis). A major problem with such diagnostic criteria is that it is necessary to wait for the child's literacy to be impaired before the problem can be addressed. Much research in the field of dyslexia has been conducted in terms of psychological constructs such as phonology, memory and automaticity. However, even those branches that have attempted to provide perspective by moving away from language areas can reveal only so much about the underlying nature of dyslexia. An improved understanding of the biological root(s) of dyslexia might not only allow clearer theoretical understanding and earlier diagnoses, but also better informed courses of remediation.

A difficulty facing theorists is that the various implicated brain systems have diverse targets and complex effects, often working cooperatively in skill learning and execution (Doyon *et al.*, 2003; Ullman, 2004). Therefore, experiments in most areas can only provide vague clues as to the affected anatomical structures. One of the 'cleanest' methods of isolating cerebellar function is to examine classical eyeblink conditioning (EBC). Steinmetz and colleagues (2001) note that "As a test of associative learning, the conditioned eyeblink response is a direct measure of motor learning. It does not require operationalization, nor is it generally affected by mood or intention. Thus, it is relatively free of confounding variables." (p. 231). Studies inactivating the anterior interpositus nucleus show that the cerebellum is essential for EBC (even in some unconventional set-ups, for example where the unconditioned stimulus is non-somatosensory (Rogers *et al.*, 1999)) and that cerebellar and related brainstem circuitry are sufficient, at least for short delay conditioning (for a review see Hesslow & Yeo, (2002)). Therefore, a finding of disrupted EBC in any given group would strongly indicate cerebellar abnormality.

3.8.1. Classical conditioning

Classical conditioning was famously reported by Pavlov (1927), who noticed dogs salivating to a stimulus that normally predicted food, even on occasions when it did not. In that example, the Unconditioned Stimulus (US) is the presentation of food, it naturally produces the reflexive Unconditioned Response (UR), here salivation. The conditioned stimulus (CS) can be completely arbitrary, for example, the sound of a bell. If the CS repeatedly occurs before the US, Conditioned Responses (CRs) will emerge. These are responses resembling the unconditioned reflex, which occur in response to the CS even in the absence of the US. In eyeblink conditioning, the UR is a blink of the eyelid. The US is most often a puff of air directed onto the cornea from ~10mm. The conditioned stimulus is generally an auditory tone and the optimal conditioned response is an eyeblink in anticipation of the US, timed to produce maximum closure as the air is delivered. The standard paradigm is delay conditioning. Here the CS (e.g. tone) will begin before the US (e.g. airpuff) but overlap with it so that the two co-terminate. Typically the ISI (inter-stimulus-

interval, between onset times) is short (e.g. 400ms in human adults). Trace conditioning is similar, the crucial difference being that the stimuli do not overlap, that is the CS terminates before US onset. Trace CS-US onset intervals are typically longer, around 1100ms in human adults.

Classical conditioning is considered a form of learning because the animal must learn that the CS predicts the US in order to produce CRs. Therefore learning is considered to have taken place if CRs are produced following presentation of the CS (if they are exhibited sufficiently frequently and other reasonable explanations for blinks before the US having been excluded). Generally the time window in which a response can be labelled a CR is large enough for there to be notable variation in CR timing, consequently another aspect of the learning in EBC is the fine-tuning of CR timing, so that CRs provide maximum protection to the eye from the US.

Much of the EBC research pointing to the cerebellum has used rabbit models and the conditioning of the nictitating membrane response (NMR).. The nictitating membrane sweeps across the rabbit's eye from nose to temple and is in addition to the external eyelids. However, the conditioning process and circuitry of the rabbit NMR are thought to have commonalities with EBC in other animals (Rogers *et al.*, 2001). There is certainly much evidence consistent with this assertion provided by patients with cerebellar lesions (Daum *et al.*, 1993; Gerwig *et al.*, 2003; Gerwig *et al.*, 2004) and by human imaging experiments (Ramnani *et al.*, 2000; Schreurs *et al.*, 2001). The situation is more complicated, especially in human participants, when the ISI is longer (e.g. > 550ms), or when a trace paradigm is used rather than a delay paradigm. In such cases there is more scope for conscious control of blinking and a strong possibility that learning is modulated by other brain structures, particularly the hippocampus (e.g. Steinmetz, (2000); Weible *et al.*, (2003)). Indeed it has been proposed that "...the hippocampal component of the eyeblink conditioning task becomes dominant when cerebellar LTD[2] is impaired." (Takatsuki *et al.*, 2003, p. 17).

3.8.2. Clinical research applications

EBC has been used previously to investigate dyslexia, but also schizophrenia, ADHD, fetal alcohol exposure, autism, and OCD (Coffin *et al.*, 2005; Hetrick *et al.*, 2004; Sears *et al.*, 1994; J. A. Tracy *et al.*, 1999). Coffin and co-workers compared controls with three separate experimental groups, these were children with either dyslexia, ADHD or fetal alcohol exposure, noting that externally the three groups share some behavioural characteristics. These researchers report that those with ADHD acquired CRs similarly to the control group but in contrast dyslexics and those who had suffered fetal alcohol exposure produced very few signs of learning (Coffin *et al.*, 2005).

[2] long-term depression

A study of classical conditioning in dyslexic adults (Nicolson *et al.*, 2002) produced intriguing heterogeneous results. Young adults with dyslexia were compared with age and IQ matched control subjects (13 participants in each group). A corneal airpuff (80ms duration) was paired with a co-terminating 800ms auditory tone, providing a 720ms ISI. Three dyslexic subjects showed no conditioning at all, while the group as a whole showed significantly worse temporal "tuning" of conditioned responses and significantly reduced habituation of the orienting response to the tone CS. Only 15% of the dyslexic group showed a normal pattern of conditioning. Mainstream research on EBC has established that the cerebellar interpositus nucleus is essential for CR acquisition. Debate continues as to the importance of cerebellar cortex for CR acquisition, although there is a strong suggestion that it is responsible for the CR's temporal tuning (Christian & Thompson, 2003; Dimitrova *et al.*, 2002; Garcia & Mauk, 1998). Specifically, it seems that without involvement of cerebellar cortex, CRs are generated too early in the trial to be considered adaptively timed (Bao *et al.*, 2002). Interpretation is complicated here by the length of the ISI, the greater the delay between the tone and the airpuff, the greater the opportunity for hippocampal, striatal or cortical involvement in the process. The 720ms interval between CS and US employed by Nicolson and colleagues could in principle have supported recruitment of these extra-cerebellar regions.

The study of EBC in this thesis aimed to verify whether there was still timing impairment in dyslexic adults' CRs when the ISI was reduced to 400ms (as predicted by Nicolson *et al.* (2002)), or indeed whether conditioning in dyslexic subjects was more severely impaired (as suggested by Coffin *et al.*, (2005)).

3.9. Issue 3: Learning and consolidation

Despite the specific reference to language acquisition in any mainstream definition of developmental dyslexia, it is undoubtedly in some sense, a learning disorder (known as a "specific learning disability" in the US and as "specific learning difficulties" in the UK, (Nicolson *et al.*, 2002)). Reading is not an innate ability but clearly and painfully a skill that must be acquired through considerable practice, exposure and instruction. As outlined earlier in this chapter, there is reason to suspect that the learning difficulties involved are not confined entirely to the literacy domain but could be rather general, reaching as far as balance and classical conditioning.

By adulthood it could be expected that any lesser skill performance deficits in dyslexia, for example those sometimes seen in balance as opposed to nonword reading, might well be very subtle. The cerebellar/automaticity framework does not expect that motor deficits are typically as pronounced as phonological deficits or that learning is impossible in dyslexia, but simply that it requires more time and effort. Therefore there is a need to aim research at the learning process itself, to highlight differences between dyslexic and control participants that may be undetectable in simple performance tests, and to elucidate precisely at what stage(s) in the learning process these differences originate. From this rationale, the third study in this thesis

aimed to investigate the behaviour of dyslexics and controls in a motor skill acquisition task.

One complicating factor in the research of the neural substrates of motor learning is that the various research groups involved adopt differing methodologies. Different tasks will inevitably yield different patterns of brain activity. Doyon and Benali (2005) have recently put forward a model of the brain circuits underlying the learning of motor skills. At the heart of this model is the distinction of two types of task: "the first measures the incremental acquisition of movements into a well-executed behaviour (motor sequence learning, MSL), whereas the second tests our capacity to compensate for environmental changes (motor adaptation, MA)." (p. 161). The authors propose that well-learned skills are represented in cortico-striatal or cortico-cerebellar circuits according to whether they fall into the MSL or MA category respectively, and that this dissociation of contributing subsystems is "...most apparent during the slow learning phase (i.e. automatization) when subjects achieve asymptotic performance, as well as during reactivation of the new skilled behaviour in the retention phase." (Doyon *et al.*, 2003, p. 252). This view is consistent with a lot of empirical evidence (Imamizu *et al.*, 2000; Karni *et al.*, 1995; Kleim *et al.*, 2004; Sakai *et al.*, 2002; Shadmehr & Holcomb, 1997; Toni *et al.*, 1998; J. I. Tracy *et al.*, 2001).

As well as different tasks, motor learning research is clouded by the different stages that skill acquisition goes through. It is common to distinguish between three stages, and these three are nicely illustrated by the experiments of Korman *et al.* (2003). Put briefly, stage one is characterized by rapid gains in performance whenever a novel behaviour is executed repeatedly. Stage two is often called consolidation and refers to latent learning progressing over several hours following initial training. During consolidation the skill becomes more specific. The expression of consolidation gains is modulated by sleep (Korman *et al.*, 2003; Walker *et al.*, 2002). Performance gains in stage three are more modest and require prolonged practice. In stage three the skill becomes yet more specific to the practiced pattern. For example it becomes less transferable from one hand to the other. At this third stage the skill is becoming automatic. The task employed by Korman *et al.* falls into Doyon and Benali's (2005) MSL category rather than the MA category.

Nicolson and Fawcett (2000) examined the long term learning of a keyboard spatial task in adolescent dyslexics and controls. The task (based on the pacman game) involves elements relevant to both of Doyon and Benali's (2005) MSL and MA skill classifications. Nicolson and Fawcett report that their dyslexic participants performed the task less well than controls overall, in terms of both speed and accuracy. Performance deficits were evident at the start of the experiment and they endured to the end. However, there was no difference between the groups in their rates of learning, nor in the strength of their automatization of the task. The dyslexic impairment in quality of performance and automatization was interpreted as consistent with the CDH of dyslexia and more problematic for the phonological and magnocellular theories.

Motor learning has long been associated with the cerebellum. For example, a PET study reported widespread cerebellar activation when participants were learning a sequence of finger presses by trial and error (Jenkins *et al.*, 1994). This procedure was later replicated using subjects with dyslexia as a test of the CDH of dyslexia. Nicolson *et al.* (1999) report that activation of the right cerebellar cortex was lower for the dyslexics than for controls when learning a new sequence.

However, many other brain structures are also involved in motor learning, for example Wu *et al.* (2004) observed that execution of untrained motor sequences was associated with activation of primary motor cortex, premotor cortex, parietal cortex, inferior frontal gyrus, prefrontal cortex, supplementary motor area (SMA), pre-SMA, cingulated cortex, basal ganglia, insular cortex and cerebellum. The pattern of brain activity on the same task after training was similar. It seems that two facets of a task might make cerebellar involvement more prominent or critical, error feedback (Doya, 2000; Jenkins *et al.*, 1994; Karni *et al.*, 1995; Toni *et al.*, 1998) and the need for specifically timed responses (Green *et al.*, 1999; Sakai *et al.*, 2002; Wu *et al.*, 2004).

Given the evidence of both motor performance deficits (Fawcett & Nicolson, 1995) and of more general automatization abnormalities (Moores *et al.*, 2003; Nicolson & Fawcett, 2000) in dyslexia, the present study aimed to further clarify at what points those with dyslexia differ from controls in terms of their learning and motor skill. The method used was drawn from Korman *et al.* (2003) where the task was the repetition of a specific sequence of five finger-to-thumb movements. It falls firmly within the domain of motor sequence learning as described by Doyon and Benali (2005), thus presumably automatizing to cortico-striatal circuits rather than cortico-cerebellar circuits. This methodology was selected because it allows for a simple measure of consolidation learning. Korman and colleagues reported that participants achieved performance gains after a 24hr break with normal sleep following training. They attributed these delayed gains to time dependent latent learning processes. Hence, the present study aimed to examine dyslexics' and controls' immediate acquisition of a well defined skill over the course of extended practice in a single experimental session as well as the latent learning evident 24 hours after training. The learning difficulties of people with dyslexia appear to be particularly in acquiring expert, automatic performance (Nicolson & Fawcett, 1990, 2000). Difficulty in automatizing a skill would be a natural consequence of impaired consolidation processes, and so the latent learning element of this experiment was a particularly important exploratory investigation.

On the grounds of previous research (e.g. Nicolson & Fawcett, (2000)) it was predicted that there would be differences between the groups in terms of their general level of performance with controls exhibiting more competent performance overall, but also a difference in learning rates over training and/or overnight with controls learning most efficiently, on the basis of a speculation that consolidation deficits might exist before the established automaticity deficit. Although it is

expected that this motor skill acquisition task will engage the cerebellum to some extent, it is not intended as a particular test of that structure's functioning for the following two reasons: First, it does not contain the explicit error feedback or timing components that seem to provoke increased cerebellar activation (see above) and second, it falls within the category described by Doyon and Benali (2005) that is presumed to automatize to cortico-striatal (as opposed to cortico-cerebellar) circuits. Some cerebellar influence would therefore be expected early in the experiments, and not in the later stages.

3.10. Issue 4: Subgroups

Studies of dyslexia have often found considerable diversity of behaviour between individuals, in terms of their particular reading difficulties, their perceived problems and especially in their performance in non-literacy areas. Indeed, even the most resolute adherents of the phonological deficit approach have pointed out that the power of phonology in predicting reading development varies across languages (Vellutino *et al.*, 2004). This raises the possibility that there are several distinct disorders that are now termed "dyslexia" and that they all have their own causes and symptoms, with the commonality of reading difficulty. Consequently it is possible that the different theoretical approaches are all right in that they all explain different dyslexias and that the pursuit of a single explanatory architecture is a hopeless quest. On the other hand maybe a single unifying framework is still possible. For example, we have seen that the cerebellum's remit is far wider than once imagined, hence the absence of classic cerebellar signs in a given individual need not imply totally normal cerebellar function. Perhaps the areas involved in such tasks were spared (or the individual has learned to compensate for these difficulties) - but more pertinent cerebellar abnormality has still caused difficulties in reading. This could be via some other delayed skill development (e.g. articulation), which is now ostensibly normal, with no deficit apparent in everyday life but only in carefully designed experiments.

By recruiting participants to take part in studies probing all three of the areas described above, it should be possible to shed light on some of these possibilities. If it was found (for example), that some participants showed difficulties across the board but others showed problems only in literacy and phonology then there would be support for the proposition of subtypes in dyslexia, in that case perhaps a purely phonological dyslexia and a second subtype caused by some lower-level processing dysfunction, possibly cerebellar dysfunction.

3.11. Aims of this thesis

The empirical sections of this thesis are divided into three studies, with the common theme of motor learning/performance. The experimental tasks were deliberately designed and selected to test an array of non-literacy abilities towards the building up of a picture of dyslexics' strengths and weaknesses. There is a great deal of evidence for motor impairment in dyslexic children but the present work examined adults. The general aims of the project as a whole are listed here.

1. Balance and postural control. To assess the balance skill and postural control of a sample of dyslexic adults and control participants and thereby shed light on the fundamentality of automaticity deficit in dyslexia.

2. Classical eyeblink conditioning. To examine classical eyeblink conditioning in a sample of dyslexic adults and control participants as a direct test of cerebellar motor learning. Enduring abnormality in this cerebellar learning into adulthood would suggest that this is close to the heart of the disorder rather than an associated developmental delay.

3. Motor skill acquisition. To investigate motor skill acquisition in adults with dyslexia and control participants in order to better describe the learning process in dyslexia. Again, any differences in learning behaviour still evident in adults are likely to be an illuminating fundamental characteristic of their disorder.

4. Co-morbidity. To establish the incidence of signs of ADD in the sample, and the effects of the inclusion or exclusion of participants with ADD tendencies.

5. Assess the range and prevalence of motor problems in dyslexia. The three studies above address a very wide range of motor functions in order to discover whether any motor deficits in dyslexia were restricted to certain types of task or whether they could be seen across the board. In addition, it is important to consider whether all participants show motor problems or whether any between group differences are the result of just a few seriously impaired individuals.

6. Assess within participant consistency of motor performance. If the prevalence of motor impairment is, say, 50% in each study, are the same individuals impaired in each experiment, or do some fall down in one area and others in a second area? In other words, for given individuals is motor impairment observable across the board or sporadically? If there is a pattern of strengths and weaknesses that emerges in the dyslexic group, how could this inform thinking on the biological level of explanation?

4. Balance and postural control

4.1. Introduction

As discussed in the introductory chapter, balance is a major issue in dyslexia research because balance experiments formed the foundation of the automaticity deficit hypothesis (Nicolson & Fawcett, 1990). It is further investigated here because existing replications have been varied in outcome and perhaps even more varied in methodology. It is traditional when examining automaticity to use a dual-task paradigm, to see whether performance of a secondary task interferes with simultaneous performance of the primary (balance) task. This design can reveal a lack of automaticity in the primary task that is normally masked by "conscious compensation" (see section 3.4). With the emergence of the cerebellar deficit hypothesis of dyslexia, balance has increased its theoretical significance since the cerebellum is known to be directly involved in postural control.

There were three modes of experimentation, all with single and dual-task conditions. They will be described separately, with experiments 1, 2 and 3 being the sensory organisation test (SOT), the adaptation test (ADT) and heel-to-toe balance respectively. Most consideration is given to experiment 3 since this is directly comparable to the body of existing research on balance and dyslexia beginning with Nicolson and Fawcett (1990). The secondary tasks employed were counting, slow choice reaction task and fast choice reaction task (CRT). These will be described in more detail in a later section (section 4.2.5.). Although the SOT, ADT and heel-to-toe experiments are to be reported separately, in reality data collection was mingled, with all balance tests carried out in a single session in the following order:

> SOT conditions 1-6 (two trials of each of C1 and C2, three trials of C3 to C6); ADT; heel-to-toe; heel-to-toe+bindfold; counting task calibration; SOTC1+counting (2 trials); ADT+counting; heel-to-toe+counting; CRT practice; heel-to-toe+slow CRT; heel-to-toe+fast CRT; SOTC1+slow CRT (2 trials); SOTC1+fast CRT (2 trials); ADT+slow CRT; ADT+fastCRT.

The heel-to-toe test is intended as a replication of Nicolson and Fawcett's (1990) earlier research, whereas it is hoped that the SOT and ADT sections will throw more light on this diverse area of balance and postural stability.

4.2. Method

4.2.1. Participants

4.2.1.1. Participant recruitment

Forty participants were recruited for the balance experiments, twenty for each of the control and experimental (dyslexic) groups. 15 Dyslexic participants were recruited

after responding to emails or telephone calls asking for research participants, these participants had previously been assessed for dyslexia in the University's Psychology Department and indicated an interest in research. Of the remaining 5 participants, two were recruited having previously participated in research projects in the same department, and 3 were personal acquaintances of the first author. Controls were recruited less formally through acquaintances of the experimenter and other researchers in the department. For all experiments reported in this thesis, ethical approval was obtain from the Department of Psychology Ethics Committee (University of Sheffield) prior to testing.

4.2.1.2. Inclusion criteria

To participate in the study, subjects were required to have a full scale IQ above 98 as measured by the WAIS-III (Wechsler, 1998) or a short form thereof. It was required that the control group had no known learning difficulty or particular reason to suspect that they might have. They were also required not to show a dyslexic profile through the psychometric testing in this study. To exclude borderline cases, members of the dyslexic group all scored an Adult Dyslexia Index (ADI)[3] of at least 2.5 out of four in their Sheffield University dyslexia assessments (Nicolson & Fawcett, 1997). Some of these criteria, and screening for alternative causes of postural stability strength/weakness, were addressed by interview.

4.2.1.3. General group descriptions

Table 4.1 gives a general description of the participants. Standard deviations (in brackets) are given below means. The large standard deviation for ages in the dyslexic group is due to two older participants aged 34 and 41. The larger proportion of males in this experiment reflects the general dyslexic population. The IQ data for the dyslexic group are actually based on only 19 participants because subtest scores were not available for the twentieth participant. His full-test full-scale IQ score was 110. For the remaining 39 participants the displayed IQ scores are derived from a short form comprising the following subtests: Picture Completion, Vocabulary, Similarities and Block Design, two subtests from each of the Performance and Verbal domains. All participants also undertook the Digit-Symbol Coding and Digit Span subtests, but these were not used in the group comparison table below since they were part of the ADI selection criteria.

[3] See appendix 1 for more on ADI scores.

Table 4.1. General descriptive data on the participants
(Standard deviations in parentheses)

Group	Age (years)	IQ (short form WAIS-III)	Male n	Female n
Dyslexic	21.8 (5.67)	120.1 (13.0)	14	6
Control	21.4 (1.53)	121.0 (16.2)	13	7

4.2.2. Additional psychometric testing

This section outlines all data collection except for the experimental balance and distracter tasks. Items falling in this psychometric category typically took between 1 hour and 1 hour 15 minutes to complete in total, including IQ tests already mentioned. Generally balance tests took place in the same session, with the exceptions of those dyslexic participants whose psychometric data had been recently collected by the department in diagnostic assessments.

4.2.2.1. Interview.

Written consent was obtained from all participants prior to testing. Before giving consent, participants read a brief description of the experiments and were given the opportunity to ask questions. They were also informed of their right to withdraw freely at any point. Following this a short interview was conducted to collect background information on each participant primarily aimed at obtaining knowledge of possible confounds, such as an estimate of alcohol consumed in the preceding 24 hours. For more information on the interview see appendix 3.

4.2.2.2. Brown ADD.

After the interview, each participant was asked to respond to the "Ready Score" Brown ADD Scale (Brown, 1996). This is a series of 40 statements (see appendix 2 for some examples) that are to be rated "never", "once a week or less", twice a week" or "almost daily" according to how often they describe the participant. The overall score obtained can be subdivided into 5 components: Activation, attention, effort, affect and memory. Six members of the dyslexia group and one control participant produced scores in the ADD "highly probable" range, the analyses are reported with and without these 7 participants.

4.2.2.3. Nonsense word reading (NWR).

The nonsense word reading test was given as in all dyslexia assessments at the Sheffield University Department of Psychology (for full diagnostic method see Nicolson and Fawcett, (1997)). The passage used is taken from Finucci *et al.* (1976), it consists of normal and nonsense words and is 96 words long in total (including 32 nonsense words). The participant is simply asked to read the whole passage through

out loud and the experimenter records the time taken (rounded up to the next whole second) and number of errors made.

4.2.2.4. WORD spelling.

The spelling test from the Wechsler Objective Reading Dimension (Wechsler, 1993) was administered as in all the ADI dyslexia assessments. This is a relatively simple test with the maximum spelling age of 17 years being attributed to anyone scoring more than 41 out of 50. The first 15 items were not administered and full credit was given. There is no time restriction in this test. Each item is dictated 3 times by the tester, the second of these is as part of a sentence to provide context for the word in question. Raw scores rather than spelling ages were used in the analyses in this thesis.

4.2.2.5. Dyslexia adult screening test (DAST).

Three simple tests were administered from the Dyslexia Adult Screening Test (Fawcett & Nicolson, 1998). They are described here in the order that they were given during the experiments. (1) One Minute Reading. Here a participant is given a list of 120 common words of increasing difficulty to read aloud as quickly as possible. The score used is the number of words read correctly in a minute. (2) One Minute Writing. The participant is asked to copy a short passage from a sheet of paper in front of them. This is to be done as fast as possible while maintaining accuracy and legibility. The basic score is the number of words completed with small penalties for spelling and punctuation errors. (3) Two Minute Spelling. The tester dictates a list of 32 words that are increasingly difficult to spell. Each word is given only once and they are given at the maximum speed that the participant appears able to cope. The score used is the number of correctly spelled words in the 2 minute time limit, with an additional 8 points for simple un-dictated words.

4.2.3. Interview outcomes

All participants were asked the same set of questions following a structured interview format and through a friendly conversational style. Generally the interview responses showed no problems, the most notable exceptions are discussed here (there is more detail in appendix 3).

On vestibular and orthopaedic impairment the most severe case was of one control participant who suffered a twisted pelvis in childhood. When questioned after the experiments he recalled no particular problem other than that standing for a long time can be painful. His condition does not slow down his movements.

On participation in sports it seems that if anything the dyslexic group were more active than the controls, which strengthens any finding of dyslexic deficit in these balance experiments.

4.2.4. *Psychometric testing results*

A table giving detailed descriptive data on psychometric test scores is below.

Table 4.2. Additional psychometric data on the participants

	Dyslexic (s.d.)		Control (s.d.)		*t*	p
Vocabulary	12.32	(1.70)	15.15	(2.41)	4.22	< 0.001
Similarities	13.16	(2.65)	12.10	(2.15)	1.37	ns
Digit Span	8.95	(1.93)	12.25	(2.90)	4.16	< 0.001
Picture Completion	12.21	(2.30)	10.65	(2.94)	1.84	ns
Digit-Symbol Coding	8.37	(1.46)	12.50	(2.59)	6.10	< 0.001
Block Design	13.37	(2.73)	13.30	(2.75)	0.08	ns
Brown ADD Score	53.10	(17.04)	34.60	(14.27)	3.72	< 0.001
NWR time (s)	84.11	(30.96)	44.25	(9.85)	5.48	< 0.001
NWR errors	11.79	(3.75)	4.65	(2.08)	7.40	< 0.001
WORD Spelling	38.68	(2.98)	45.75	(1.77)	9.05	< 0.001
DAST Reading	81.68	(20.95)	118.30	(11.68)	6.79	< 0.001
DAST Writing	29.63	(5.41)	36.10	(3.61)	4.41	< 0.001
DAST Spelling	28.58	(3.49)	35.95	(2.87)	7.22	< 0.001

(all 2-tailed, independent groups t-tests)

The pattern of results shown in the table is as expected. Note that higher scores reflect higher level of performance with the following exceptions: NWR measures and ADD measures (in the latter a higher score represents more ADD tendencies). 2-tailed independent t-tests revealed significant between group differences ($p < 0.001$) for all tests with the exception of three WAIS subtests (picture completion, block design and similarities) where the dyslexic group scored slightly better, the difference nearing significance in the case of picture completion ($p = 0.07$).

4.2.5. *Secondary cognitive tasks*

Three forms of secondary distracter task were employed: Counting, slow choice reaction time (CRT) and fast CRT as outlined below.

4.2.5.1. Counting

Following the procedure of Nicolson and Fawcett (1990), this task was calibrated with the aim of making the task of equivalent difficulty when performed alone. Therefore there was a graded sequence of counting tasks (in order of ascending difficulty):

(a) Up in 2s from zero, (b) Down in 1s from 100, (c) Down in 2s from 100 (default), (d) Up in 3s from 100, (e) Down in 3s from 200, (f) Up in 7s from zero, (g) Down in 7s from 300.

The participants were asked to perform one of these at a time (beginning with the default), as fast as they could, until they produced close to 18 correct operations in 30 seconds. The version of the task in which they performed closest to this ideal became the version they would perform in all dual task experiments. In practice no participant counted at level 1 or 2. During 'counting' dual task experiments the experimenter played a series of tones simply to provide a standard pace for the counting. There was one tone every two seconds so the test rate though rigid was normally slower than the rate at which participants had performed their test task during calibration. The participants were simply asked to perform the particular balance test as they had done before, with the minimum movement possible and to say the next number in their sequence each time they heard a beep. Responses were tape recorded although some of this data is missing due to some participant's speaking too quietly to be picked up clearly by the machine used.

4.2.5.2. Slow and fast CRT

The speeds of presentation in the omission Choice Reaction Task were one stimulus per second and two stimuli per second providing the 'slow' and 'fast' CRT conditions respectively. In all other ways these two were the same. The stimuli were a series of tones identical to those used in the 'count' condition (frequency: 440Hz, duration: 0.25s) with the addition of an occasional higher frequency tone (the target, frequency: 587.23Hz, duration: 0.25s). The participants were asked to respond "yes" whenever they heard the target tone and to ignore all the other tones. Before the CRT-balance dual task experiments all participants confirmed that they could hear the difference between the two tone frequencies and were allowed one 30 second practice of each of the slow and fast tasks. In the stimuli the order of tones was randomly determined and a different stimulus track was used for each condition to prevent learning of the sequence. One in six tones was the target tone but the stimulus tracks generated were longer than needed for the experiments so the percentage of target tones heard in each experimental condition varied. However, the rate of the need to process information remained constant.

4.2.6. Secondary task baseline performance

4.2.6.1. Counting

The difficulty of each participant's counting task was adjusted with the explicit aim of equivalent baseline counting performance between groups. The aim was to find a counting task that the participant could perform at a rate of around 18 operations in 30 seconds. At the end of the calibration phase the mean dyslexic counting rate (for each individual's specific task) was 18.2 / 30 seconds (min = 14, max, 23, s.d. = 2.38), the mean rate for the control group was 18.7 / 30 seconds (min = 15, max = 24, s.d. = 3.01). The difference was not significant (t = 0.52, n.s.).

4.2.6.2. CRT

This was performed at two speeds (1 presentation/second and 2 presentations/second). Each participant was allowed 30s practice of each mode

before they were combined with the balance tests. There was no significant difference between percent correct for each group when the slow version was practiced. The dyslexics were significantly worse ($t = 2.88$, $p < 0.01$) when practicing the fast CRT but still achieved > 90% correct.

4.3. Experiment 1: Sensory organisation test (SOT)

This test was administered using posturography monitoring equipment borrowed from DDAT (Dyslexia, Dyspraxia and Attention deficit Treatment centres - www.ddat.co.uk). The equipment is used to assess DDAT clients' progress with an exercise program designed to improve cerebellar function (Reynolds *et al.*, 2003), so it was expected that persons with dyslexia who had not undergone the DDAT intervention would show significant difficulty in the SOT.

4.3.1. SOT test procedure

Participants stand in a cuboidal chamber and are fitted with a harness in case they begin to fall. The walls to their front, left and right are able to move as one with the ceiling. The floor is able to move separately. The floor is also pressure sensitive, enabling the computer running the SOT to track a participant's centre of gravity, and hence knowing their height, it can also calculate sway angles. The SOT uses "sway-referencing" to match movements of the floor and/or the wall-ceiling combination to a participant's spontaneous movements, thereby removing cues to orientation. People primarily use visual, vestibular and proprioceptive information to judge their orientation, the latter being predominantly from the angle of the ankle. So if when one leans forwards or backwards, and the floor moves an equivalent amount in the same direction; then the angle of the ankle remains constant and that cue to sway is removed. Similar reasoning follows for the movement of the visual world (walls and ceiling). Hence, if sway referencing is applied to the floor or visual surround, then the proprioceptive or visual cues (respectively) can be said to have been removed or disrupted and therefore stable posture will be reliant on good use of the remaining accurate cues. The sensory organisation test comprises 6 conditions:

Condition 1 (C1) – all cues available
Condition 2 (C2) – blindfold
Condition 3 (C3) – sway-referenced visual surround
Condition 4 (C4) – sway-referenced floor
Condition 5 (C5) – blindfold and sway-referenced floor
Condition 6 (C6) – sway-referenced floor and ceiling

The computer running the test returns a score out of 100 (the equilibrium score) indicating the participant's stability in a given trial, with 100 being no movement. Each trial lasts for approximately 22 seconds. As standard, two trials were administered in each of the first two conditions and three in each of the remaining trials. Additional trials were administered where there was some irregularity, from apparent failure to understand instructions to equipment malfunction. After this standard administration of the SOT, condition 1 was combined with each of the three secondary tasks, yielding nine SOT conditions in total.

4.3.2. SOT results

In the six standard conditions of the SOT there was little difference between the groups, with the first (simple balance) and second (blindfold balance) closest to separating the groups (see table 4.3). However, the controls averaged higher stability scores in all conditions. When the tests were repeated excluding the 7 participants with high ADD scores the general pattern remained - stabilities were consistently but non-significantly higher in the control group.

Table 4.3 Results of the standard SOT conditions.

Equilibrium scores (max score = 100)

	C1	C2	C3	C4	C5	C6
Dyslexic						
mean	93.95	91.37	93.89	84.89	62.00	63.74
st. dev	*3.61*	*5.17*	*2.88*	*8.24*	*11.36*	*22.20*
Control						
mean	95.43	93.50	94.85	88.55	66.30	64.40
st.dev	*1.12*	*2.09*	*2.98*	*5.69*	*7.09*	*15.24*
t	1.76	1.71	1.02	1.62	1.43	0.11
p	0.09	0.10	ns	ns	ns	ns

A 1-within, 2-between ANOVA was undertaken with SOT condition as a within subject factor (levels: Single(C1), Blind (C2), Count, Slow, Fast) and GROUP and SEX as between subjects factors. There was a significant effect of Condition $F(4,140) = 7.74$, $p<0.001$. The main effect of GROUP did not reach significance $F(1,35) = 3.15$, $p = 0.09$. There were no further significant effects. It should be noted that the controls showed greater mean stability in all of these conditions. Between groups t-tests for these 5 conditions indicated a significant difference in the slow condition ($t = 2.38$, $p<0.05$), but there were no further statistical differences between groups. Exclusion of those with ADD tendencies weakened the between group difference in the slow condition ($t = 1.81$, $p = 0.08$), while the non-significant results in the count and fast conditions remained.

It will be noted that the later (more difficult) conditions produced much larger standard deviations. It seems likely that this is due to ceiling effects in the earlier conditions which would have prohibited large variability in C1-C3.

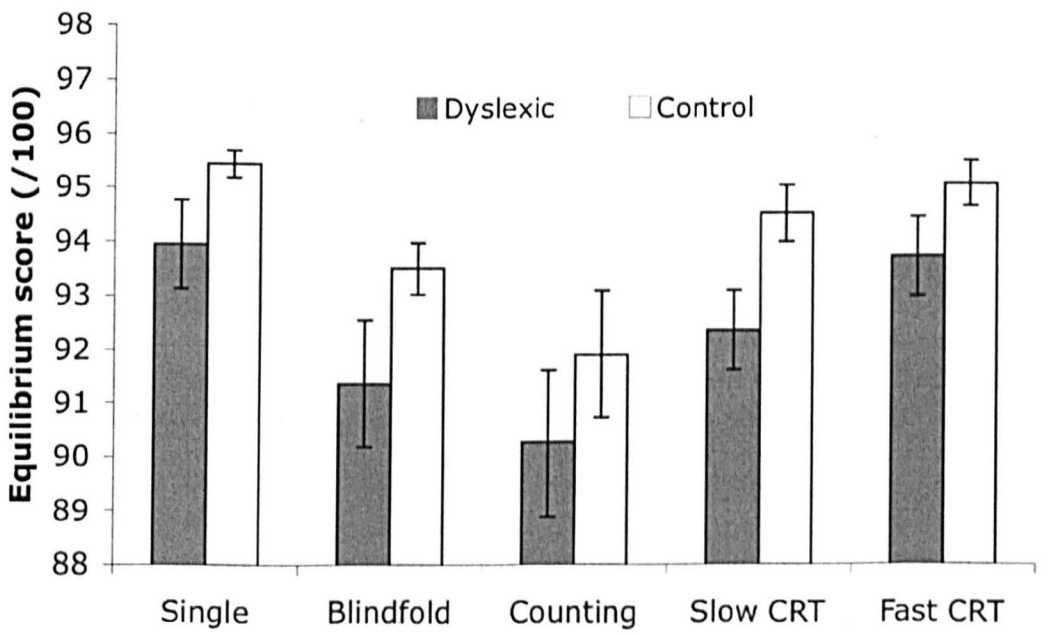
Table 4.4 SOT results including dual-task conditions

Equilibrium scores (max score = 100)

	Control	Dyslexic	*t*	p
Single				
mean	95.43	93.95	1.76	0.09
st. dev	*1.12*	*3.61*		
Blindfold				
mean	93.50	91.37	1.71	0.10
st. dev	*2.09*	*5.17*		
Counting				
mean	91.90	90.25	0.92	ns
st. dev	*5.27*	*6.08*		
Slow CRT				
mean	94.50	92.35	2.38	< 0.05
st. dev	*2.37*	*3.27*		
Fast CRT				
mean	95.05	93.70	1.60	ns
st. dev	*1.90*	*3.25*		

Figure 4.1. SOT results in single and dual-task conditions.

Bars represent +/1 standard error throughout this thesis.



4.3.2.1. Counting

When balance during counting was measured using the SOT there was no significant difference between the percent correct[4] mathematical operations performed by the members of each group, although the dyslexic group did make slightly more errors. This was true whether or not the high ADD scorers were included.

4.3.2.2. CRT

When the choice reaction tasks were paired with the SOT, there was a trend for dyslexics to make more errors than controls during the fast version ($t = 1.82$, $p = 0.08$) but not the slow version. This mirrors the practice session. There was no significant difference between groups without those who showed signs of ADD.

4.3.2.3. Individual analyses

Individual analyses were conducted to examine what proportion of the dyslexic group was showing balance problems in those conditions where group level analyses had indicated dyslexic deficit, and to investigate whether certain individuals consistently struggled with balance throughout the SOT conditions.

The individual analyses were conducted using effect sizes, which were obtained by finding the difference between an individual's score and the control group mean score on a given test, and then dividing that difference by the standard deviation of the control group's scores. Hence the effect size is in standard deviation units with signs always arranged so that a positive effect size indicates performance 'better' that the control group's performance and negative effect sizes indicating 'worse' performance. An effect size of -1 would describe performance 1 control standard deviation below the control mean and so on. Throughout this thesis, an effect size of -1 or worse is taken to denote "impaired" performance on that test. Comparison of effect size magnitudes between tasks gives an index of which tasks prove the most problematic for the adults with dyslexia.

At least one participant in each group showed impairment in each SOT condition, and therefore it cannot be claimed that balance impairment as measured using the SOT is exclusive to dyslexia. There were four conditions that produced impairments for more than a third of the dyslexic group. None of the conditions caused such widespread difficulty in the control group. These problematic conditions were conditions 1, 4 and 5 of the standard SOT and the slow CRT condition, with 40%, 37%, 37% and 50% of the dyslexic group (respectively) performing at least 1 standard deviation below the mean control level. Examination of the data implies that there was substantial overlap of those impaired in the dual-task slow CRT balance test and those impaired in the undistracted conditions.

---

[4] (Number of correctly performed operations / number of tones), each tone being an invitation to respond.

4.3.3. *SOT discussion*

As predicted there was a general trend for the Sensory Organisation Test to prove more difficult for participants with dyslexia. Also in line with the predictions it should be noted that the one condition to show a strong difference between the dyslexic and control groups was one of the dual-task conditions, suggesting that a secondary task is necessary to distract conscious attention from postural control and thereby provide a purer and more sensitive test of (automatized) balance. Sensitivity seems to be a key issue here, with both groups averaging equilibrium scores above 90 out of 100 on all conditions where the floor was stable, including the dual-task conditions. It seems likely that these results are full of ceiling effects, rendering the emergence of statistically significant between group differences very unlikely. The relatively easy to maintain posture adopted for this test (a normal standing position with feet side by side) is comparable to that used by Franck Ramus and colleagues who also saw little evidence of between group differences (Ramus *et al.*, 2003b). Further comparison of this and other balance studies follows later in this chapter and includes comment on postural variations.

Regardless of whether or not balance deficits exist in dyslexia, it was surprising to find such a high level of postural stability in the dyslexic group as a whole, as assessed by the SOT, since this equipment is used as an indicator of progress for people with dyslexia, dyspraxia and attention deficit (hyperactivity) disorder in their remediation programs at DDAT centres. However it should be noted that these participants were students and therefore not representative of many people who seek treatment for dyslexia.

The relatively high incidence of dyslexic balance impairment in SOT conditions 4 & 5 (compared with other conditions) is in line with general findings at DDAT centres (R. Rutherford, personal communication). The common factor in these conditions is the removal of somatosensory feedback by sway-referencing of the floor, leaving increased reliance on visual and vestibular information, or solely vestibular information in the case of condition 5. It should be born in mind that the results of this SOT experiment are not strong, nevertheless it is noteworthy that these vestibular-demanding conditions seem most troublesome in dyslexia, since it is this mode of information (of the three) that is likely to be most heavily dependent on the cerebellum.

4.3.4. *Conclusions*

The SOT provided a weak indication of balance deficits in dyslexia, overall it appeared to be too easy a test with few participants showing any notable difficulty. However, there was an indication that some members of the dyslexic group were poor at making use of vestibular cues to balance.

4.4. Experiment 2: Adaptation test (ADT)

This test was administered using the same DDAT posturography monitoring equipment as the SOT. It is a dynamic test in that it can be used to assess changes in postural stability with learning opportunities. Also because (in contrast to the SOT) the ADT involves movements generated purely by the posturography equipment.

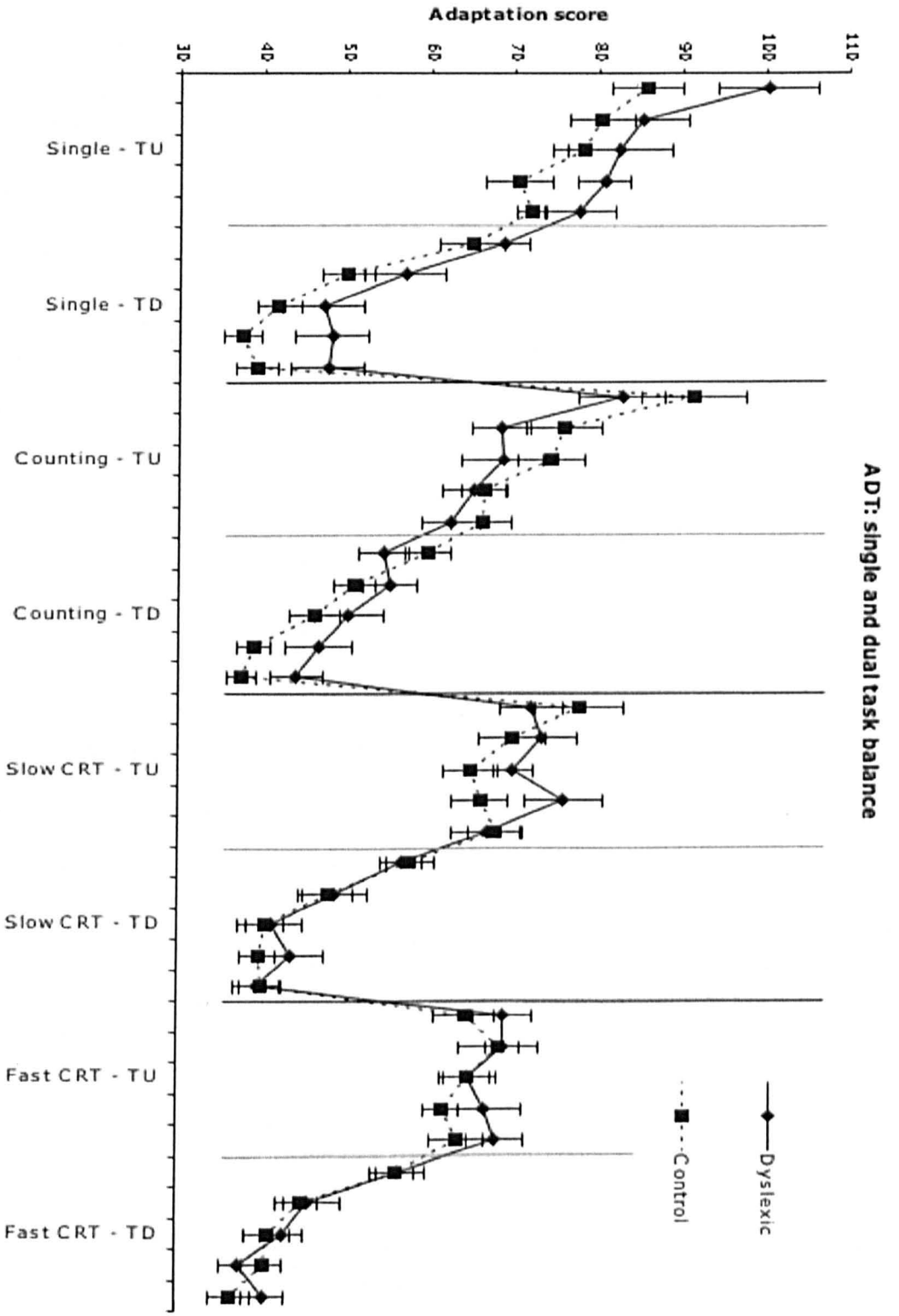
4.4.1. ADT test procedure

The equipment produces 5 consecutive movements of the platform in the toes up direction (throwing the participants backwards and disrupting balance), followed by 5 in the opposite (toes down) direction. The whole test takes a little under 70 seconds. There is no movement of the visual surround. The participants are simply instructed to stand as still as possible and warned that there will be a series of sharp, sudden movements of the floor. Again the computer generates a summary ("Adaptation") score for each trial (each movement of the floor) but contrary to the SOT, here a larger score indicates more movement. It is expected that participants will learn to inhibit their reflexes and adapt their responses to the floor's movement, so that by the fifth identical movement of the floor, their destabilisation is considerably less than it first was. So there is an opportunity to study gross motor adaptation here. In addition, the extent of movement in the very first trial will serve as an overall indicator of postural control/motor coordination. Motor adaptation learning and postural control specifically, both engage cerebellar processing (Dow & Moruzzi, 1958; Doyon & Benali, 2005; Holmes, 1917). Therefore, from the perspectives of the automaticity deficit and cerebellar deficit theories of dyslexia, it is expected that there will be significantly better adaptation in the control group than in the dyslexic group. Furthermore, it is predicted that there will be greater instability in the dyslexic group in general, as measured by the amount of movement seen in the first trial, indeed, a simple measure of postural stability has proven an effective part of a range of dyslexia screening tests (Fawcett & Nicolson, 1996). The adaptation test was undertaken four times, once as described and a further three times with the counting, slow CRT and fast CRT secondary elements.

4.4.2. ADT results

Three types of output are considered: (i) The gradients of the eight learning slopes, two for each of the four conditions (toes up and toes down). (ii) The actual adaptation scores for each group at the first and last trial of each slope (larger scores reflecting greater destabilization). (iii) The cost of switching back to the toes up task from the more recent toes down task at the beginning of an adaptation condition (excluding the first condition). It is worth recalling at this stage that all participants undertook their 4 adaptation tests in the same order: Single, count, slow, fast.

Fig 4.2 Adaptation scores from each trial for both groups
Including standard error bars (TU = toes up, TD = toes down).



(i) Generally the two groups produced similar profiles, both improving dramatically in the first two tests and less in the later tests. The only significant difference between the groups' learning rates was in the second (toes down) half of the 'count' test. While asked to perform their counting task during the toes down adaptation test, dyslexics showed a much more shallow learning gradient (less learning) than did the control group ($t = 3.25$, $p < 0.01$ or $t = 2.47$, $p < 0.05$ when those with high ADD scores were excluded from the analyses). This finding is confirmed by a 1 within 1 between ANOVA with TRIAL (toes down 1-toes down 5) as the within subjects factor and GROUP (dyslexic/control) the between subjects factor. There was a strong overall learning effect, TRIAL $F(4,80) = 15.42$, $p < 0.001$ but no main effect of GROUP, $F(1,20) = 0.26$, n.s. Crucially the GROUP by TRIAL interaction was significant, $F(4,80) = 2.80$, $p < 0.05$. This interaction was decomposed by way of the line of best fit analysis mentioned above, which clearly shows faster improvement in the control group. The pattern of results was preserved when data from participants with high ADD scores were excluded (TRIAL $F(4,76) = 14.1$, $p < 0.001$; GROUP $F(1,19) = 0.05$, n.s.; GROUP by TRIAL $F(4,76) = 2.55$, $p < 0.05$).

Further analyses of variance were carried out (including all participants) to confirm the general learning effect for the other three toes down tests and all four toes up tests. The effect of GROUP was never significant, indicating no overall difference in stability between dyslexics and controls in the adaptation test. There was a significant effect of trial for all but the final toes up test. There was also a significant GROUP by TRIAL interaction for the toes up test with simultaneous slow CRT, $F(4,96) = 5.16$, $p < 0.01$. Examination of the graph again suggests that the learning is most substantial in the control group, however this interpretation does not stand up when the interaction was examined through the line of best fit analysis, since there was no significant difference between the groups' learning gradients ($t = 1.49$, n.s.).

(ii) Apart from the actual learning, it is also interesting to examine whether at any particular point the overall stability of a group deviated most from the other. I will describe two instances. (i) The very first trial. For the first time the participants were subjected to the sudden movement of the platform, the dyslexics as a group appeared to be more seriously destabilized than controls ($t = 1.95$, $p = 0.06$, or $t = 2.19$, $p < 0.05$ without high ADD scorers). (ii) By the end of the 'count' adaptation test the control group seemed to have developed a better response to the platform's movement than the dyslexic group ($t = 1.84$, $p = 0.08$, or $t = 1.24$, n.s. without high ADD scorers) despite the opposite ordering at the start of the test. So in both these instances there was a trend towards statistical significance. Only at one point was there a between groups difference that reached significance at the 5% level. Control performance was superior in the first (un-distracted) ADT test in the fourth toes down trial ($t = 2.19$, $p < 0.05$).

(iii) Overall the toes up tests appear to have provoked much more movement than the toes down tests, this can be explained fairly intuitively in terms of the shape of

feet. During the course of the experiments this switch of adaptation test task from easy (toes down) to hard (toes up) was required 3 times. It is striking that the change of task seems to have been more costly for the control group than for the dyslexics. When looking at the relevant last and first trials where these 3 switches were demanded the GROUP x TASK interaction was found to be significant in the first instance, $F(1,37) = 5.78$, $p < 0.05$, but not in the two later instances ($F(1,33) = 1.78$, $p = 0.19$ and $F(1,38) = 1.23$, $p = 0.28$ respectively, the same pattern is observed without the participants who scored highly on the ADD scale: $F(1,31) = 4.55$, $p < 0.05$, $F(1,29) = 1.00$, n.s., $F(1,31) = 0.30$, n.s.). Indeed in the third, the groups' scores were very similar with the slight trend in the opposite direction. It should be noted that in this third case the two tests were undertaken consecutively, whereas there were breaks at the other two task switching times that included other tests and a chance to rest.

4.4.2.1. Counting

During ADT testing the percent correct[5] mathematical operations performed by the members of each group was significantly different, with the control group showing the advantage ($t = 2.89$, $p < 0.01$, or $t = 2.71$, $p < 0.05$ without high ADD scorers).

4.4.2.2. CRT

When the slow and fast CRTs were performed during the ADT, the dyslexics made significantly more errors than controls in the fast condition ($t = 2.39$, $p < 0.05$, or $t = 2.11$, $p < 0.05$ without high ADD scorers) as they had done in the fast CRT practice. This is a particularly noteworthy finding because at this point in the experiments the participants have practiced this very simple task several times. In spite of this, the dyslexic participants still produced significantly less correct responses than the controls in this dual-task setup despite control performance being close to ceiling in the practice session. There was no CRT performance difference between the groups in the (earlier) slow condition ($t = 1.47$, n.s., or $t = 1.50$, n.s. without high ADD scorers).

4.4.2.3. Individual analyses

Individual analyses were carried out here using effect sizes in the way described for the SOT results (section 4.3.2.3). The three variables examined were those that appeared able to discriminate between groups: Performance at trial 1, performance at the last trial of the counting-toes down section and learning gradient during the counting-toes down section. 30%, 41% and 47% of the dyslexic group showed impairment on these three measures (respectively), compared with 15%, 17% and 11% of the control group. The elements under examination (general postural control and motor adaptation while distracted) appear to be unrelated, since, of the 6 dyslexic participants impaired at trial 1 only 1 is also impaired at the end of the counting-toes down section. Two of these six were impaired in their learning rates during the counting-toes down section.

---

[5] (number of correctly performed operations / number of tones), each tone being an invitation to respond.

4.4.3. ADT discussion

In common with the SOT results obtained using the same equipment, these ADT results hint at a general impairment in stability for the dyslexic group, who seem to have been more seriously destabilised by the first movement of the platform. Beyond that finding, again in common with the SOT, there were no differences between the groups without the complication of the secondary (counting) task. The impaired adaptation of the dyslexics' responses while counting suggests that improvements they showed in the ADT-alone condition may have been due to a more conscious strategy making approach than those seen in the control group.

Finding (iii) above that switching to a more difficult condition appeared more costly for the control group has precedent in a study of long-term learning in dyslexic children (Nicolson & Fawcett, 2000). Here the task was modelled on the pac-man game but several manipulations were made to the task throughout the course of the experiment. Reversal of the required route, for example, appeared not to influence performance speed for participants with dyslexia, but to reduce completion times in the control group. As motor skills are learned to higher and higher levels of skill and automaticity they tend to become less adaptable, and less transferable (Korman *et al.*, 2003). Perhaps that is what has happened here, the less finely-tuned dyslexic performance has retained more adaptability and therefore is less hindered by task alterations.

4.4.4. Conclusions

Motor adaptation learning clearly involves many areas of the brain. According to Doyon and Benali (2005), it draws heavily on two loops, through striatum and cerebral cortex and through cerebellum and cerebral cortex, with the latter becoming more dominant as learning progresses. Given the range of data suggesting cerebellar dysfunction in dyslexia from other experimental domains (Nicolson *et al.*, 2001) it seems reasonable to suggest that the dyslexics' impaired unconscious adaptation, seen here in the counting condition, is also attributable to cerebellar abnormality. This is not universal in the dyslexic group, but instead is evident in 41-47% of participants with dyslexia.

4.5. Experiment 3: Heel-to-toe balance

4.5.1. Heel-to-toe test procedure

Experiment 3 was designed to augment the balance paradigms used by Fawcett and Nicolson (Fawcett & Nicolson, 1992; Nicolson & Fawcett, 1990) with automatic balance monitoring employed by other more recent studies (Moe-Nilssen *et al.*, 2003; Poblano *et al.*, 2002; Ramus *et al.*, 2003b). The Polhemus "FASTRAK" equipment (www.polhemus.com/fastrak.htm) was used to record subjects' stabilities when maintaining the heel-to-toe position. This is standing one foot in front of the other with heel touching toe in a straight line and with arms outstretched. Participants were asked to stand like this for a minute at a time moving as little as possible. The Polhemus equipment consists of a central hub connected to a laptop computer via USB. A "transmitter" box is also connected to the hub and this serves as a reference point on the ground. Up to four sensors can also be plugged in to the hub. The software records the 3D co-ordinates of all sensors in use at a rate of > 50 times per second. In these experiments two sensors were used, one attached to the forefinger of each outstretched hand. This follows Nicolson and Fawcett (1990) where similar balance experiments were video recorded and much of the variation between subjects was in the form of arm movements (generally up and down). Thus in the present study arm movements indicative of balance difficulty were objectively tracked.

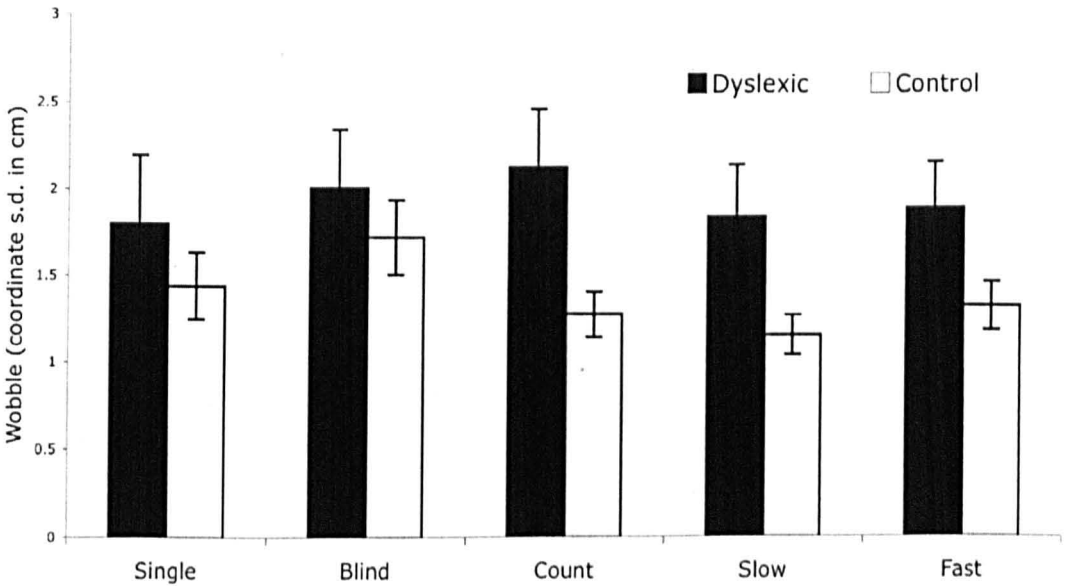
4.5.2. Heel-to-toe balance results

Between 3000 and 3500 sets of co-ordinates were analysed for each 60 second balance test. These were split into 7 time bins, each 500 readings long, with the seventh being anything after 3000. Bins 1 and 7 were not used in the analysis on the assumption that data from the beginning and end of the tests would be particularly uncharacteristic of performance as a whole. Artefacts were removed and the dependent variable "wobble" calculated as the mean of the standard deviations of the remaining 5 data bins. This analysis was calculated separately for each axis. 3D results were obtained by taking the mean of the x, y and z results in each case. All participants completed all the heel-to-toe tests but for three dyslexic participants the equipment appears not to have worked effectively and so their data was omitted. For the count condition the equipment failed for another two dyslexic participants, however, usable z axis data was recovered for one of these.

Mean performance data are given in figure 4.3. It may be seen that the dyslexic group wobbled more in every condition. A 1-within, 2-between ANOVA was undertaken with balance condition as a within subject factor (levels: Single, Blind, Count, Slow, Fast) and GROUP and SEX as between subjects factors. Here the main effect of CONDITION was not significant [$F(4,124) = 0.93$, n.s.] However there was a significant effect of GROUP, with the dyslexics balancing significantly less well overall [$F(1, 31) = 4.33$, $p < 0.05$], and a significant interaction between CONDITION and SEX, [$F(4,124) = 3.54$, $p < 0.01$] with females significantly more stable than males in the single ($t = 2.13$, $p < 0.05$) and fast ($t = 2.86$, $p < 0.01$)

conditions but not in the Blind, Count and Slow conditions (all *ts* n.s.).[6] Independent groups t-tests showed that the amount of 3D movement of the dyslexic and control groups was significantly different in the count and slow dual task conditions (with superior balancing in the control group) but not in the other three conditions (see final column of table 4.5).

Figure 4.3. Heel-to-toe balance in single and dual task conditions



Heel-to-toe balance data given thus far have been based on all three dimensions (that is mean movement in the x, y, and z axes). The x axis represents movement from left to right. The y axis represents movement from front to back (toe to heel). The z axis measures vertical movement. It may be seen that the pattern of between group differences in dual-task conditions is preserved in all 3 dimensions (table 4.5).

A series of t-tests was carried out to determine the significance of any between group differences for each condition. As can be seen from the table, there are significant differences between the groups, but only in the dual task conditions. In all conditions, the mean wobble for the dyslexic group was higher than that for the control group.

---

[6] The ANOVA was repeated omitting the seven participants showing signs of ADD (two of these were participants for whom heel-to-toe data were unavailable so only five were actually excluded at this point). The pattern of significant results was preserved, with the following exception, the CONDITION by GROUP by SEX interaction was now significant [$F(4, 104) = 2.84$, $p < 0.05$] [GROUP, $F(1, 26) = 4.46$, $p < 0.05$; CONDITION by SEX, $F(4, 104) = 4.52$, $p < 0.01$].

Table 4.5. Heel-to-toe balance (sway) in each dimension
Group means, t-values and significance levels.
(all tests 2-tailed)

	x axis		*y* axis		*z* axis		All axes	
Single		t		t		t		t
Dyslexic	2.08	0.916	2.21	0.802	1.09	0.685	1.80	0.859
Control	1.64		1.74		0.92		1.44	
Blind		t		t		t		t
Dyslexic	2.46	1.076	2.41	0.389	1.14	0.643	2.00	0.750
Control	1.94		2.18		1.02		1.71	
Count		t		t		t		t
Dyslexic	2.62	2.557	2.41	2.091	1.30	1.866	2.12	2.642
Control	1.41	*	1.59	*	0.81	†	1.27	*
Slow		t		t		t		t
Dyslexic	2.12	1.895	2.10	2.102	1.27	1.642	1.83	2.255
Control	1.25	†	1.42	*	0.78		1.15	*
Fast		t		t		t		t
Dyslexic	2.29	1.858	2.07	1.413	1.28	2.020	1.88	1.955
Control	1.50	†	1.58		0.86	†	1.32	†

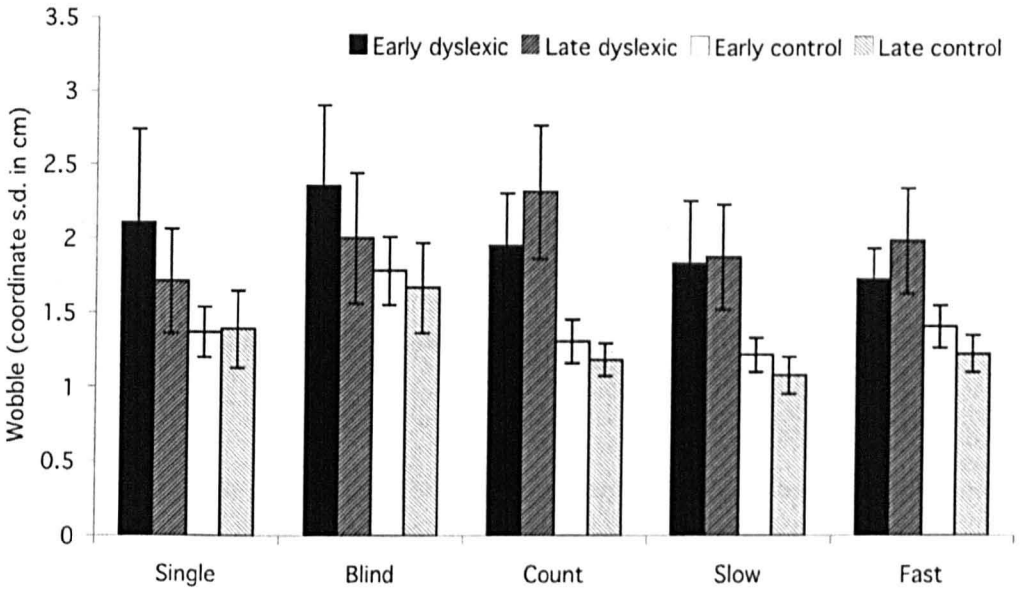
** - $p < 0.01$
* - $p < 0.05$
† - $p < 0.1$

Removal of the participants with ADD tendencies alters the pattern of significant results slightly, such that the x, y and z components of the count condition moved down one level of significance and the x component of the slow condition moved up to the 0.05 level. Significance levels for all 3D measures were unchanged.

4.5.2.1. Time effect

The balance data displayed above are based on 5 time slots during the test as described earlier. Further analyses were carried out comparing the first two of these 5 slots (c10-29 s) and the last two (c40-59s). The group means are displayed in figure 4.4, and indicate that differences between groups tended to emerge late in trials. Inferential stats are summarised in table 4.6.

Figure 4.4. Heel-to-toe balance shown early and late in the test



An interesting distinction appears to occur in that in the single task conditions the dyslexic participants seem to improve over time, whereas this improvement does not occur when under dual task conditions. By contrast there is little effect of time for controls.

Table 4.6. Group means and between group significance (p) values for sway early and late in the one minute balance test period (2-tailed t-tests, all axes).
(Symbols as for table 4.5)

		Single		Blind		Count		Slow		Fast	
Early	Dyslexic	2.10	ns	2.35	ns	1.95	†	1.83	ns	1.73	ns
	Control	1.37		1.78		1.31	(ns)	1.22		1.41	
Late	Dyslexic	1.71	ns	2.00	ns	2.32	**	1.88	*	1.99	*
	Control	1.39		1.67		1.18	(*)	1.08		1.23	(†)

As before this analysis was repeated without those participants scoring in the ADD "highly probable" range. Most significance levels were unchanged. Where there were changes, the new values are shown in brackets in table 4.6. The considerable reduction in number of participants generally seems to have weakened between group differences slightly. To investigate further, the reduction-in-wobble from early to late was calculated for each participant in all five conditions (using all axes). A correlation analysis was undertaken including these change-in-wobble measures, ADD total score, and ADD subpart scores. There was no significant relationship

between change-in-wobble and ADD composite score. The one significant wobble change – ADD correlation came from a positive relationship between improvement in the single condition and the attention sub-section of the ADD scale ($r = 0.336$, $p < 0.05$, $n = 37$). In other words, in the single condition, those who struggle to maintain attention improved most during the course of the balance task.

4.5.3. Secondary task performance

4.5.3.1. Counting

As noted earlier, the difficulty of each participant's counting task was adjusted with the explicit aim of equivalent baseline counting performance between groups. During the heel-to-toe balance tests the dyslexic group did in fact make fewer counting responses than the controls ($t = 2.95$, $p < 0.01$, without ADD participants: $t = 2.60$, $p < 0.05$).

4.5.3.2. CRT

When performed concurrently with the heel-to-toe balance test, there was no significant CRT accuracy difference between groups at either speed.

4.5.3.3. Effect size analyses

Effect size analyses (see section 4.3.2.3) were used in order to facilitate comparison between performance on the different tests (table 4.7).

Table 4.7. Between group effect sizes

	All participants		Without 5 high ADD scorers	
	Primary (balance)	Secondary (accuracy)	Primary (balance)	Secondary (accuracy)
Counting	-1.46	-1.13	-1.47	-1.04
Slow CRT	-1.38	-0.49	-2.03	-0.35
Fast CRT	-0.90	-2.47	-1.12	-2.40
Balance only	-0.42	•	-0.61	•
Balance blindfold	-0.30	•	-0.46	•

The effect sizes for dual-task heel-to-toe balance are considerable, given that the rule of thumb (Cohen, 1988) is that an effect size of -0.80 or bigger is "large". It should be noted that the effect sizes change little with the omission of the "ADD highly probable" participants and that they are all in the direction predicted by the automatization deficit hypothesis.

4.5.4. *Individual analyses*

As discussed in the introduction, one of the key issues for dyslexia and balance is the establishment of the percentage of dyslexic participants actually showing a balance problem. This can be addressed by individual effect size analyses (see section 4.3.2.3.). Looking at the effect sizes for the five balance tasks and three accuracy scores in table 4.7, we find that 80% of the dyslexic group show one or more impairment, but so do 55% of the controls. More stringent criteria were adopted where only balance performances (not secondary task performances) were used, cutting the number of tests down to five.[7] In this new analysis, 53% of the dyslexic group are impaired on 2 or more measures (as opposed to 15% of controls) and 24% were impaired on 3 or more measures (compared to 10% of controls). Incidentally, exclusion of the single and blindfold conditions from this analysis (leaving just the 3 dual-task balance tests) does not alter the pattern of impaired/unimpaired classification for the dyslexic group, but decreases the number of participants impaired in the control group. In other words, this study's general finding of specific difficulty with dual-task balance for dyslexic adults is reinforced. To conclude from these individual analyses it can be said that balance impairment is 2 to 3 times more prevalent in the dyslexic group (24%-53%) than in the control group (10-15%).

4.5.5. *ADD*

Throughout this results section, analyses have been presented with and without those dyslexic participants exhibiting ADD tendencies. However, the mean Brown ADD score was higher for the dyslexic group as predicted by Wimmer and colleagues (1999), and therefore, in line with one of the key issues for dyslexia research (co-morbidity and subgroups) identified in the introductory chapter, the relationship between balance and ADD was investigated further. In terms of 'impairment' in score on the ADD scale (as indicated by an effect size of –1 or worse), 50% of the dyslexic group were impaired and 20% of the control group. However, of those 9 dyslexic group members who were impaired at least twice on balance, only 5 were also impaired on ADD. A Chi-square analysis confirmed there was no association between balance impairment and ADD impairment (chi-square = 0.202, n.s.). Correlations between balance measure and ADD effect sizes were all non-significant (all $rs < 0.2$), in other words there was no evidence that those with ADD tendencies were more likely to be particularly good or bad at balance. In summary, at least in this dyslexic sample, the issue of mild ADD appears orthogonal to that of balance impairment.

4.5.6. *Heel-to-toe discussion*

The main aim of experiment 3 was to extend the heel-to-toe balance paradigm to adults, using an objective method of balance assessment. Crucially, if significant

[7] At this point the number of dyslexic participants was also reduced from 20 to 17 since heel-to-toe balance data were not available for these three participants due to technical failure as mentioned above.

deficits were found (compared with age- and IQ-matched controls), then this would constitute evidence of disorder, rather than developmental delay in this group. A second issue was to assess the prevalence of any balance difficulties in the population sampled.

The major finding is that there was an overall main effect of group in the heel-to-toe balance performance. Additionally, as predicted from the Nicolson and Fawcett (1990) and Fawcett and Nicolson (1992) studies, this difference was attributable almost entirely to performance in dual task conditions. Furthermore, in addition to significant difficulties in balance in the dual task conditions, the dyslexic adults showed significant difficulties in performance on the secondary counting task. This indicates that the dual task balance difficulties cannot arise solely from a trade-off between primary and secondary tasks strengthening the findings. It has therefore been established that there are indeed significant problems in balance in adult dyslexic participants. The results support the findings of balance deficits in dyslexic children (Fawcett & Nicolson, 1992; Moe-Nilssen *et al.*, 2003; Nicolson & Fawcett, 1990; Stoodley *et al.*, 2005; Yap & van der Leij, 1994) and extend them to dyslexic adults.

The variety of existing finding is this area will now be addressed, with particular reference to the adults study of Ramus and co-workers (Ramus *et al.*, 2003b) and Wimmer's research with Austrian children. Ramus and his colleagues tested dyslexic students and controls on several balance tests but found no evidence of balance impairment. As noted earlier, it is likely that the tests used were not in the appropriate range of sensitivity to reveal differences, especially given that the fundamental test was two foot, side by side, balance rather than heel-to-toe. Nonetheless, Ramus *et al.* did find considerable heterogeneity in their dyslexic group as did the present study (and previous Sheffield studies, Fawcett and Nicolson, (1999); Nicolson and Fawcett, (1994a)). In the present study, though there were a significant proportion of the dyslexic sample who had balance difficulties (24-53% depending on criterion), there were certainly some who showed no evidence of balance problems (20% in the present sample had no balance or secondary task at risk scores in the heel-to-toe experiments), and it is clear that there were also controls (10%) who showed strong evidence of balance problems.

A further issue of interest is the relationship between ADHD, balance and dyslexia. It was suggested earlier in this thesis (section 3.7.1.) that the Austrian children tested by Wimmer and colleagues (Raberger & Wimmer, 2003; Wimmer *et al.*, 1999) may not have been directly comparable to a typical group of children diagnosed with dyslexia in the U.K. In particular the focus on reading speed without reference to accuracy in German speaking countries would be likely to have had an effect. Nonetheless, there is clearly an association between dyslexia and higher scores on the Brown ADD score, as indicated by the between group effect size of –1.30 even though there appears to be no association between ADD and balance within the

dyslexic group.[8] It is possible that this high scoring is due to some cross-over of symptoms of the two disorders. In particular, one fifth of the ADD score is derived from memory items, which persons who have been diagnosed as dyslexic would be likely to score highly on. Indeed, short digit-span contributed towards diagnosis in many of the present participants.

The differences between early and late balance periods are worthy of discussion here. The original hypothesis (Nicolson & Fawcett, 1990) was that the dual task conditions prevented conscious compensation. The improvement of the dyslexic participants from early to late in the balance minute (but with no such improvement under dual task conditions) is directly consistent with this hypothesis. In addition, the greater effect in the later stages of dual-task tests may be due to an inability to unconsciously regain a stable posture once wobbling has begun. Note that all participants were competently holding the required posture at the start of each test. One final point that should be made relating to the time effect in heel-to-toe balance is that it helps to explain the weaker findings in other experiments. Not only was the posture dictated by the SOT test easy to maintain (being similar to the Ramus *et al.*, (2003b) posture), but each trial lasted little more than 20 seconds, which would certainly not have been long enough to detect the dyslexics' balance deficit in these, more sensitive, heel-to-toe tests. Furthermore, test lengths were shorter in the studies of the Wimmer (Wimmer et al., 1999) and Ramus (Ramus et al., 2003b) research collaborations, being 30 and 40 seconds long (respectively) as opposed to 60 seconds in the present study.

Experiment 3 was set up to investigate whether replication of the original (Fawcett & Nicolson, 1992; Nicolson & Fawcett, 1990) balance studies with adult dyslexics would reproduce the results originally obtained. The pattern of results was consistent with those obtained earlier, in that significant between group differences were found in the dual task conditions but not in the single task conditions. There is also support for other studies of balance. There does appear to be something of an association between dyslexia and a higher ADD score (as suggested by Wimmer *et al.* for German-speaking children), but unlike the Wimmer studies (Raberger & Wimmer, 2003; Wimmer *et al.*, 1999) there was no association between balance and ADD score. There was also some support for the Ramus (Ramus *et al.*, 2003b) position, in that (taking a relatively stringent criterion for risk) only 9 of the 20 dyslexic group were at risk on balance. The results therefore reconcile the apparent differences in the literature, and suggest further potential developments. The distinction made

[8] The Brown scale does not address hyperactivity directly and therefore we refer to the Brown scores as ADD rather than ADHD. The scale is made up of five component scores (activation, attention, effort, affect and memory). The only component related to hyperactivity is "activation", with higher scores reflecting greater slowness in getting started on tasks. The dyslexic group scored significantly higher than the controls (2 tailed $t = 2.80$, $p < 0.01$), and therefore showed hypo- rather than hyper-activity.

between early and late balance may be a particularly promising avenue for further research.

4.6. General conclusions from balance and postural control.

Several conclusions can be drawn at this stage. First, it is important to use tests of appropriate sensitivity when investigating the balance impairments of people with dyslexia, which are often subtle. The heel-to-toe test used in experiment 3 seems most appropriate, at least for adult participants. Variations in posture and procedure could account for the discrepant published results. Second, the group of participants were diverse with respect to their balance and postural control. This reinforces the proposition that the dyslexic population is highly heterogeneous and consequently it is advisable to pursue a broad research program that will build up a wide-ranging profile of strengths and weaknesses for each participant. Finally, the results are consistent with the original Nicolson and Fawcett (1990) findings, and therefore provide support for the automaticity deficit hypothesis of dyslexia.

5. Classical eyeblink conditioning

5.1. Introduction

Classical eyeblink conditioning provides a valuable clean probe of cerebellar motor learning (Steinmetz *et al.*, 2001), and therefore an excellent opportunity to test the cerebellar deficit hypothesis of dyslexia. For example, lesions of the cerebellar interpositus nucleus consistently block conditioned response acquisition while contributions from other brain areas appear less crucial (Christian & Thompson, 2003; Garcia & Mauk, 1998; Steinmetz, 2000). For a full introduction to the literature see the introductory chapter. The present study investigated three key issues: First, would the dyslexic group still show impairment in the tuning of their conditioned responses (CRs), despite the reduction in the CS-US interval from a previous study (Nicolson *et al.*, 2002), as predicted by the cerebellar deficit hypothesis? Second, would the dyslexic group produce less conditioned responses than the control group as observed in dyslexic children by Coffin *et al.* (2005). Third, would members of the dyslexic group exhibit slowed habituation to the tone CS (persistent alpha responses) as observed by Nicolson *et al.* (2002).

5.2. Method

5.2.1.1. Participants

Matched groups of dyslexic (n=18) and control (n=16) adults were recruited (see table 5.1). The dyslexics were recruited by telephone or email from a list of those having registered an interest in research participation at the time of their diagnoses. As in the previous study, they all met the dual criteria of (i) IQ exceeding 98 and (ii) Adult Dyslexia Index (ADI) of 2.5 or above in the diagnostic assessment, indeed many took part in both studies. The maximum ADI score is 4 and it is derived from 4 equally weighted components, (i) childhood diagnosis, (ii) nonsense passage reading, (iii) the WORD spelling test and (iv) WAIS-III profile (Nicolson & Fawcett, 1997). More info on ADI scores is given in appendix 4. Control participants were also recruited from a panel of previous research participants with three exceptions. These three were referred to the experimenter by existing control group members. All controls underwent the same set of tests as the dyslexic participants without showing evidence of dyslexia.

Table 5.1 General descriptive data on the participants

	Age (years)	IQ (short form WAIS-III)[9]	Gender (m/f)	Handedness (R/L)
Dyslexic	20.83	122.89	13/5	14/4
(s.d.)	(1.38)	(12.06)		
Control	21.50	120.50	11/5	13/3
(s.d.)	(1.41)	(16.01)		

5.2.1.2. Psychometrics

The majority of these data were collected at the time of diagnosis for the dyslexic participants, which was always after their enrolment at university. Some control participants were part of a testing panel and therefore recent psychometric data were already held for these people. Other testing was carried out on the same occasion as the experiment. The Brown Attention Deficit scale (Brown, 1996) was administered to new participants immediately before the experiment as it is not part of the dyslexia diagnostic battery. The diagnostic battery comprises the WAIS-III (or a short-form), the WORD spelling test, reading of a nonsense passage (from Finucci *et al.*, (1976)) and three subtests of the DAST, (1-minute reading, 1-minute writing and 2-minute spelling), for further detail on diagnostic testing see Nicolson and Fawcett (1997).

5.2.1.3. Procedure

All participants were given a short introduction to the procedure before being asked for written consent. Following consent they reported their handedness along with any visual or auditory problems they suffered. Six participants in each group reported some form of minor visual problem, in most cases this was short-sightedness. Three wore contact lenses but two of these removed them for the duration of the experiment. The third, who preferred not to remove them, produced many more CRs than the group average and so it is assumed that conditioning was not inhibited. More information on the interview is given in appendix 4.

5.2.2. Eyeblink conditioning

During the experiments eyelid movement was measured by recording the amount of infrared reflecting from the participants' retinas. The apparatus[10] comprises a headset on which the infra red recorder and the airpuff delivery tube are mounted next to each other on a flexible arm. The auditory stimuli are presented via separate headphones. Each participant was seated in front of a television and was fitted first

[9] This short-form comprised the Vocabulary, Similarities, Picture Completion and Block Design subtests.

[10] Supplied by San Diego Instruments. See http://www.sd-inst.com/prod_eyeblink.htm for further details of the equipment used.

with the headset, adjusted so that it would not move during the experiment, and then with the headphones. The infra red recorder was positioned using the software's "digital scope" facility to ensure that eyeblinks were being detected, with the air supply tube being directly above the probe. When all the apparatus was in place, participants were reminded of their instructions[11] and a video was played (silently) to maintain participant alertness.

5.2.2.1. Baseline measurements.

A number of "micro-sessions" were used to obtain baseline measurements and calibrate the apparatus. During all sessions, white noise was delivered via the headphones to mask background laboratory noises and it relented only during presentation of the principle stimuli. The first session was an acclimatization period (ACC) lasting 191 seconds. Eyelid movement was recorded for 2 seconds (equivalent to 1 conditioning trial) at intervals of 5, 6, 7 or 8 seconds during this session, to give a measure of spontaneous blinking. This session also serves to allow participants time to become accustomed to their surroundings.

Next, the airpuff US was presented 10 times, on each occasion it lasted 100 ms and began 900 ms into the recorded "trial" period, which itself was always 2000 ms long. Inter-trial-intervals were varied between 20 and 28 seconds. During the first 5 presentations the air pressure was increased gradually (2,4,6,8,8 psi). The pressure was then maintained at 8 psi for the remainder of the experiment. The following 5 presentations were used to obtain a record of each participant's typical unconditioned response to the airpuff, and shall be referred to as the "CAL" (calibration) session. The positioning of the probe was adjusted during the US-alone trials according to the instant computer readout, to produce the optimal sensitivity to retinal reflection. This was normally achieved before the CAL session began. The final pre-conditioning micro-session comprised 5 presentations of the auditory tone CS (conditioned stimulus) to provide an indication of participants' responding to that stimulus when it was neutral (i.e. before conditioning). This will be referred to as the "CS" session. The tone, when presented, always began 500 ms into the 2000 ms trial period mentioned above. It always terminated at 1000 ms (simultaneously with the air when both were presented together). Every presentation of the tone was at 84dB (± 0.5dB) and a frequency (pitch) of 1kHz.

5.2.2.2. The conditioning session.

Immediately before the conditioning session ("CON") it was checked that the eye probe was positioned as it had been when the maximal CAL session response was

[11] This reminder was based on the following text: "So to summarize, all I want you to do is to sit still and watch the film. After a few minutes you will begin to experience the stimuli described above. I will tell you when the experiment is over. You are free to leave before the end if you are uncomfortable. Try not to move your head too much."

recorded. The session itself was made up of 60 trials, these ran without break except for inter-trial-intervals as described above. The session can be thought of as 6 identical blocks of 10 trials. In each block, there were 8 (paired) conditioning trials. Every 6th trial was a US-alone trial identical to those in the CAL session and every 10th trial was a CS-alone trial identical to those in the CS session. The presentations of the auditory and tactile stimuli were the same in the paired trials as in the US-alone or CS-alone trials, therefore the interval between the onsets of the two stimuli (the ISI) was 400 ms.

The conditioning session was followed by a final, 10-trial extinction session ("EXT"), identical to the previous sessions in every way except that it was made up entirely of CS-alone trials.

5.2.2.3. Debriefing

Immediately after the experimental procedures, participants were asked the following questions:

1. What did you notice about the experiment?
2. Did you think there was any relationship between the air and the beep?
3. Did you learn or notice the pattern of when there was just a beep or just air?
4. Did you feel any more bored at the end than you did early in the experiment?

Following these questions, participants were allowed to ask any questions they wanted to ask and were paid £5.

5.3. Results

5.3.1. Data analysis

5.3.1.1. Blink measurement

Blinks were measured using an infra red probe that monitors and records reflectance off the retina when positioned in front of an eye. During each 2 second trial reflectance was measured and logged every millisecond by computer. In this analysis, all responses are compared to the largest response recorded in the CAL session for the individual concerned. Therefore all figures for blink amplitude are percentages of the maximum amplitude of that largest CAL session blink. Apparent responses are only considered blinks if their peak amplitude is not below 10% of that standard blink. Along with a blink's peak amplitude, its time of onset and time of peak are also analysed. The onset time is defined as the time of the first of 4 consecutive increasing amplitude values (after the data had been statistically smoothed and filtered). The peak time is simply defined as the time when the amplitude is greatest between onsets. In addition, a whole trial is discarded if its baseline appears unstable. In other words, if the peak amplitude recorded in the initial 200 ms of the trial is more than 20% of the peak amplitude in the standardisation trial recorded in the CAL session. In general, data analysis was carried out by the author using software supplied by the manufacturers, however some additional analysis involved a program written in MatLab[12] by Mark Humphries and Jonathan Chambers of the Department of Psychology, University of Sheffield.

5.3.1.2. Response categories

CS onset was 500 ms into the trial, a blink was considered a CR if it had an onset after 650 ms and before 900 ms (the time of airpuff onset). This window was extended by 200 ms for CS-alone trials. Following standard procedure, blinks beginning between 500 ms and 650 ms were considered too fast to be CRs and more likely to be startle responses to the tone, these are labelled "alpha" responses. An unconditioned response was recorded when a blink began after US onset but within 200 ms of US onset. It was considered that later responses could not be reflexes triggered by the air. Blinks beginning before the onset of any stimuli, or more than 200 ms after the normal US onset time were classified "No Stim." (no stimulus) responses.

5.3.1.3. Data from baseline measures

In the ACC session there were no significant differences between groups in terms of the frequency or timing of spontaneous blinks (all $ts < 0.90$). The mean maximum calibration blink recorded for the dyslexic group (3024mV, *s.d. 1705*) was slightly larger than that for the control group (2658mV, *s.d. 2148*), but this difference did not approach statistical significance. During the calibration session the control group

[12] http://www.mathworks.com/products/matlab/

produced unconditioned responses more frequently than the dyslexic group (t=2.19, p<0.05), but both groups showed strong response rates (4.75 or 4.3 responses in 5 trials). The control group also blinked slightly earlier than the dyslexic group (~8ms) but this was not statistically significant.

There were no differences between the groups in their blinking in the CS session. Neither group blinked frequently. On average, in the 5 trials the controls blinked 1.13 times (s.d. = 1.54) while dyslexic participants blinked 1.11 times (s.d. = 1.37).

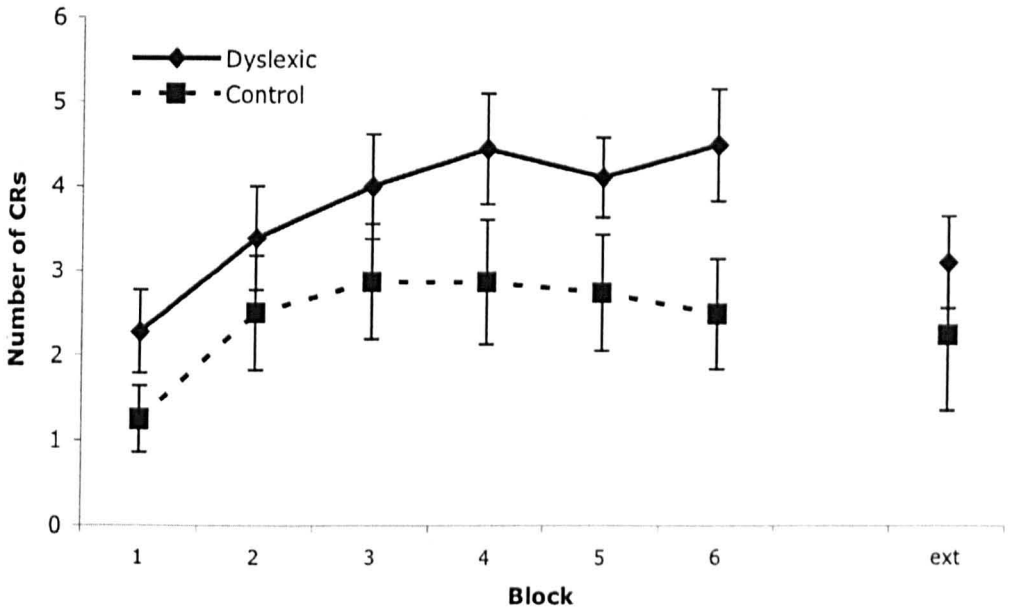
Examination of the data from conditioning session US-alone trials indicates that there was no difference in the reaction times of the two groups. Each took around 75 ms to initiate a reflexive blink on average, however, the peak of a dyslexic blink tended to be slightly later ($t = 1.74$, $p < 0.1$, 2-tailed). Block by block data show similar patterns for the two groups in terms of changes in onset times, peak times and amplitudes. There were no between group differences at any time for these variables.

5.3.2. Conditioning measures

5.3.2.1. Total CRs

The mean number of CRs produced by each participant throughout the experiment seems marginally higher in the dyslexic group (26 and 17 respectively, 2-tailed $t = 1.71$, $p < 0.1$). Although the dyslexic group appear to have produced more CRs (3.1) than controls (2.3) in the 10 EXT session trials, neither group responded frequently and the difference is not statistically significant. Figure 5.1 shows that dyslexics produced CRs more frequently at every stage of the conditioning session, although the difference is sometimes minimal and always non-significant with the exception of block 6 ($t = 2.14$, $p < 0.05$).

Figure 5.1. Frequency of CR production by each group.



5.3.2.2. Conditioning criterion

A simple test of conditioning is to set a criterion by which it is judged whether or how quickly conditioning has occurred. Here it was considered that there was good evidence of conditioning if the participant produced CRs after 20% of CS presentations in the conditioning session. The CS was presented 54 times (including 6 CS-alone presentations) and therefore participants who achieved more than 10 CRs were said to have adequately acquired the association. The criterion was passed by 14 of the 18 dyslexic participants and 8 of the 16 control participants.

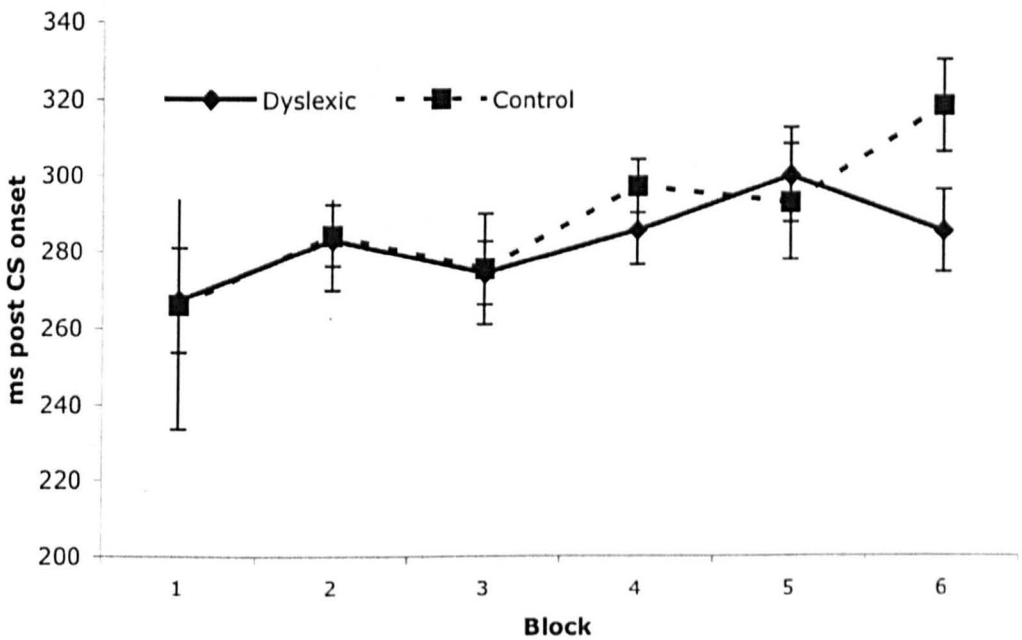
5.3.2.3. Flexibility

Somewhat surprisingly, the dyslexic group produced more conditioned responses than the control group in the final block of conditioning trials ($t = 2.14$, $p < 0.05$). In the following extinction session where all trials were CS-alone the rate of responding in the dyslexic group fell off dramatically. In contrast the reduction in response frequency was more modest in the control group, the between group difference no longer statistically significant. On a cautionary note, the dyslexic group clearly had more scope for a slowing of CR production, however it is striking that there was no significant decrease in response rate for the controls but a significant decrease for the dyslexics ($t=3.90$, $p < 0.01$).

5.3.2.4. CR timing

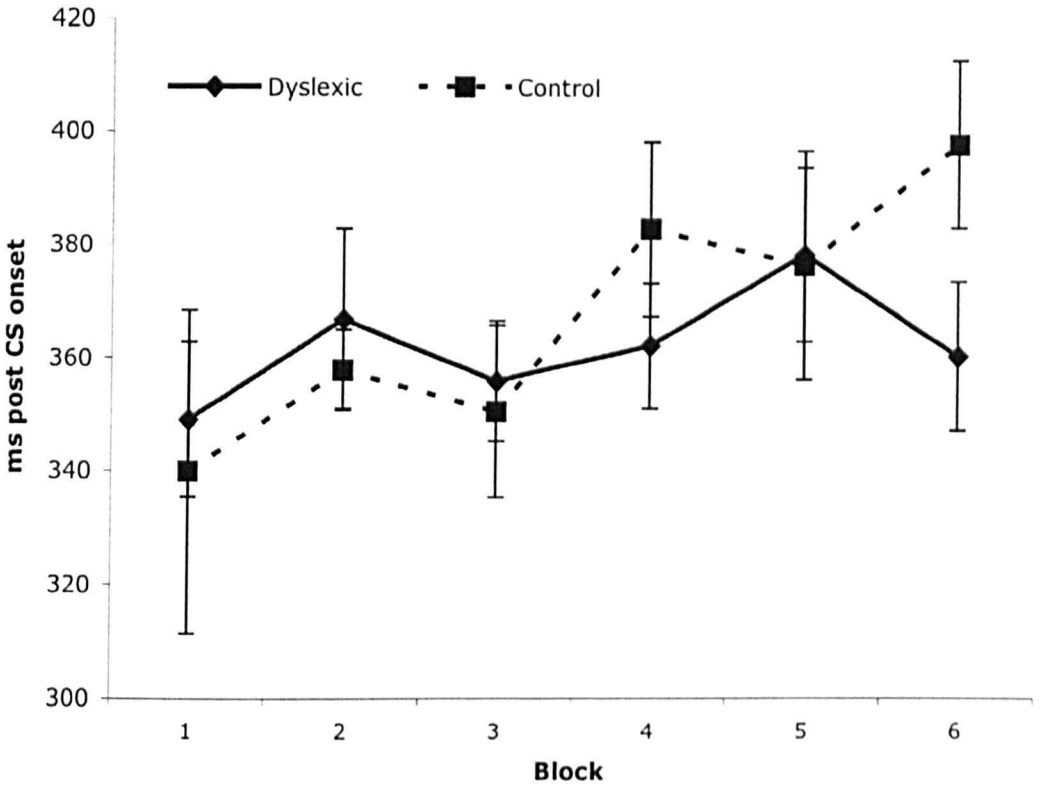
Participants who did not achieve the conditioning criterion were not included in analyses of CR timing. Given that blink responses to the unconditioned stimulus have taken in the region of 50 ms to reach their peak amplitude for each of the groups in the baseline trials of this study, it can be inferred that the ideal conditioned response will have an onset after 350 ms (timing from CS onset) otherwise the blink might be in decline before US onset. It is expected during such a conditioning session as this, that participants would "tune" their CR timing so as to minimize air to the eye. In other words, to make their CRs more like the ideal CR just described. Figure 5.2 shows the mean CR onset times for the two groups during each conditioning block. It is clear that whilst response timing is virtually identical for the two groups at first, by block 6 the mean control onset time is moving towards the ideal 350 ms mark, whereas the dyslexic group's response timing is not better than at block 2. At block 6, the response onset times of the two groups are significantly different (1-tailed $t = 2.26$, $p < 0.05$).

Figure 5.2. CR onset latencies.



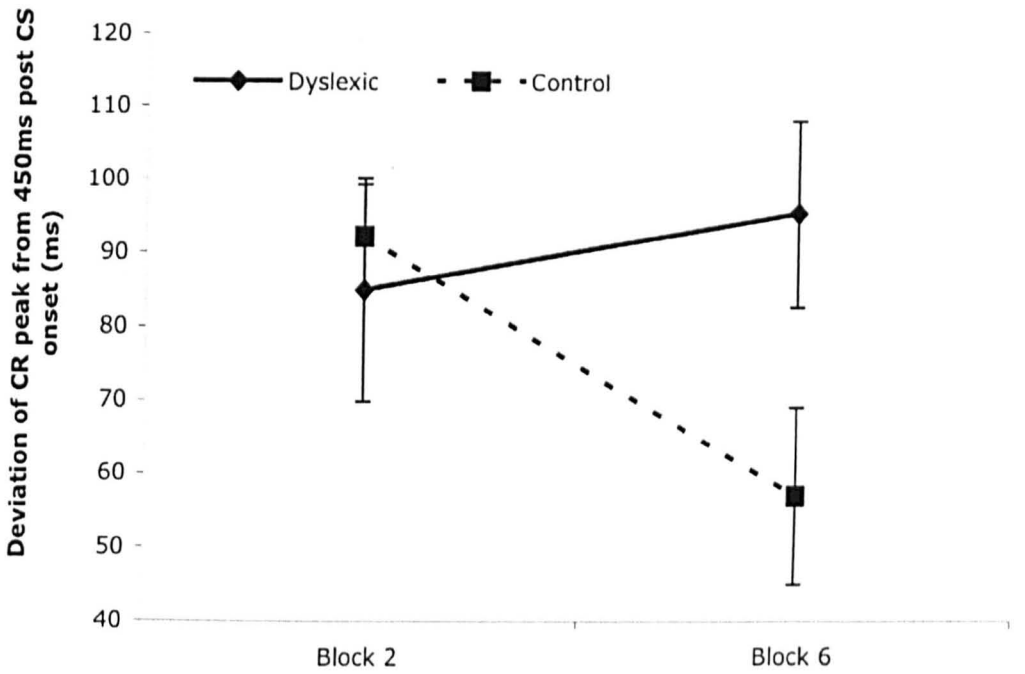
A similar pattern is evident for CR peak times (fig. 5.3), here the ideal peak was taken as 450 ms post CS-onset (half way through the 100 ms air delivery period) and the significant block 6 between group difference remains as predicted (1-tailed $t = 2.16$, $p < 0.05$), with better timed blinks in the control group (mean blink times post CR onset were dyslexic: 360.33 ms, and control: 397.79 ms, where 400 ms is the time of US onset.).

Figure 5.3. CR peak latencies



In an attempt to examine this peak tuning more specifically, peak time data from blocks 2 and 6 were compared with the ideal peak time of 450 ms to provide measures of "start error" and "end error" (fig. 5.4). Block 2 was used rather than block 1 because block 1 contained far fewer CRs. This approach clearly shows an improvement in the control group's timing that is absent in the dyslexic group. A 2 factor ANOVA with the between subjects factor "GROUP" and the within subjects factor "TUNING" (levels: "start error" and "end error") showed no significant main effects, but a significant interaction reflecting the superior timing improvement in the control group compared to the dyslexic group [$F(1,19) = 4.84$, $p < 0.05$]. However, Fisher's protected t-test showed that the timing improvement within the control group was itself not significant ($t = 0.14$, n.s.).

Figure 5.4. Temporal tuning of the CR by each group



It was mentioned under "baseline measures" above (section 5.3.1.3.) that there was a trend for the dyslexic unconditioned response to take longer than the control UR to reach peak from onset. This pattern was not found in the groups' conditioned responses with both groups taking around 80 ms from blink onset to blink peak.

5.3.2.5. Alpha responses

There was no difference between the groups in the number of alpha responses (responses with onsets less than 150 ms after tone onset) produced in any block of conditioning trials or in CS-alone trials in the conditioning session (all *t*s < 1). Furthermore, alpha responses were rare, with an average of 4.00 in the dyslexic group and 3.94 in the control group. Only three participants in each group produced 7 or more alpha responses (an average of more than 1 per block).

5.3.3. Categorization

Table 5.2 Individual results

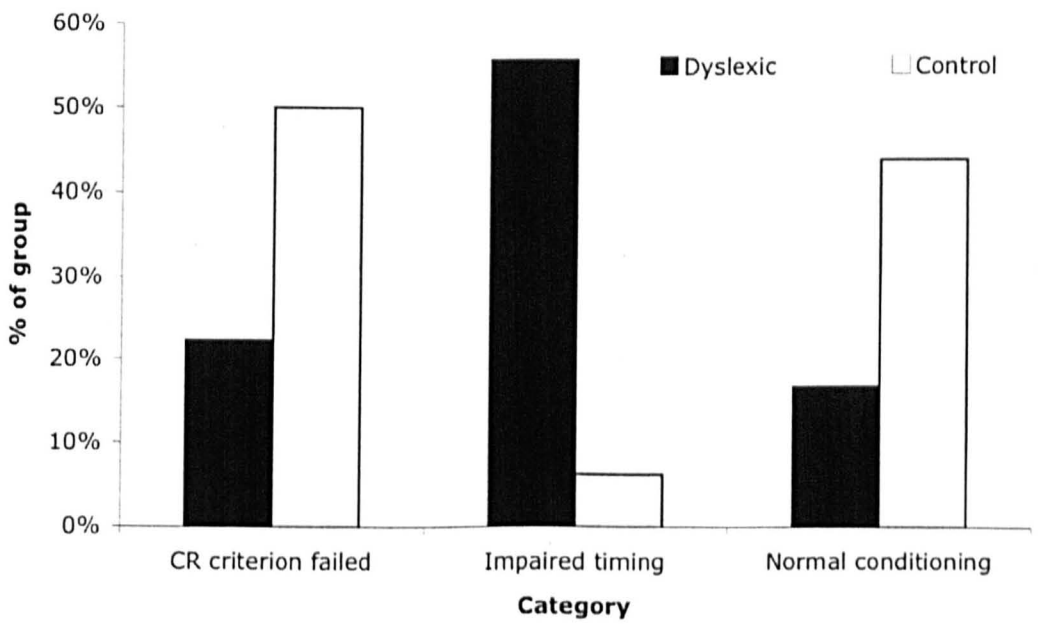
Legend: Individual results. Spelling Age = spelling age in years based on the WORD spelling test; NWR errors = number of errors made in nonsense word reading; NWR time = time taken to read nonsense word passage in seconds; Total CRs = number of conditioned responses produced in conditioning session; Block 2 peak timing error / Block 6 peak timing error = difference between mean CR peak time in those blocks and the halfway point in airpuff delivery; peak timing error reduction = change in error from block 2 to block 6. Categories: D = Did not produce CRs reliably; I = Inaccurate CR timing in final block; N = Normal conditioning; T = Final timing good but timing error increased substantially from block 2 to block 6.

Participant	Age	Spelling Age	NWR Errors	NWR Time	Total CRs	Block 2 peak timing error	Effect size	Block 6 peak timing error	Effect size	Peak timing error reduction	Effect size	Category
D1	19	11.9	18	67	8							D
D2	20	16.3	9	61	45	178.43	-4.30	101.88	-1.31	76.55	1.34	I
D3	20	11.0	16	88	27	33.50	2.92	65.20	-0.24	-31.70	-2.16	T
D4	22	12.6	19	171	6							D
D5	21	13.3	13	76	15	12.00	3.99	101.00	-1.29	-89.00	-4.02	I
D6	20	>17.0	8	65	47	152.14	-2.99	134.25	-2.26	17.89	-0.56	I
D7	21	11.9	15	90	37	114.86	-1.13	74.00	-0.50	40.86	0.19	N
D8	22	14.0	10	98	14	34.50	2.87	30.00	0.79	4.50	-0.99	N
D9	21	>17.0	11	71	35	80.00	0.61	95.57	-1.13	-15.57	-1.64	I
D10	21	14.9	11	86	30	31.00	3.05	52.29	0.14	-21.29	-1.82	T
D11	21	14.0	17	58	33	65.75	1.32	13.00	1.29	52.75	0.57	N
D12	21	16.3	13	52	29	41.33	2.53	144.67	-2.57	-103.33	-4.48	I
D13	20	15.6	9	67	29	124.80	-1.62	154.60	-2.86	-29.80	-2.10	I
D14	21	14.9	10	67	19							N
D15	24	>17.0	11	78	44	149.29	-2.84	132.57	-2.21	16.71	-0.59	I
D16	23	12.6	15	69	44	87.50	0.23	141.75	-2.48	-54.25	-2.89	I
D17	20	13.3	16	82	7							D
D18	18	11.0	16	127	9							D
D Mean	20.83	14.9	13	82	26.56	85.01	0.36	95.44	-1.13	-10.44	-1.47	
C1	23	>17.0	6	50	3							D
C2	21	>17.0	4	48	1							D
C3	24	>17.0	7	74	12	85.00	0.36	77.00	-0.58	8.00	-0.88	N
C4	22	>17.0	7	44	4							D
C5	21	>17.0	4	43	3							D
C6	23	>17.0	8	35	10							D
C7	22	>17.0	2	50	20	106.33	-0.71	19.50	1.10	86.83	1.67	N
C8	20	>17.0	1	48	31	88.83	0.17	17.25	1.17	71.58	1.18	N
C9	19	>17.0	4	42	31	68.00	1.20	37.60	0.57	30.40	-0.15	N
C10	21	>17.0	5	39	1							D
C11	23	>17.0	4	49	4							D
C12	20	>17.0	6	47	36	97.80	-0.28	98.80	-1.22	-1.00	-1.17	I
C13	23	>17.0	1	36	38	65.40	1.33	30.50	0.78	34.90	-0.01	N
C14	21	>17.0	4	45	37	126.40	-1.70	87.00	-0.88	39.40	0.14	N
C15	21	>17.0	3	70	46	99.63	-0.37	89.00	-0.94	10.63	-0.79	N
C16	20	>17.0	4	40	1							D
C Mean	21.50	>17.0	4	48	17.38	92.17	0.00	57.08	0.00	35.09	0.00	

D = Did not produce CRs reliably, I = Inaccurate CR timing in final block, T = Timing error increased, N = Normal conditioning.

The participants can be split down into three categories, as shown in figure 5.5: (i) those who fail to produce CRs with any reliability, (ii) those whose CR timing is impaired, and (iii) those who exhibit normal conditioning. Participants were only assigned to the CR timing impaired group if they passed the conditioning criterion described above. In addition they exhibited impairment of 1 effect size[13] or greater for the temporal accuracy of their CR peaks in block 6, or, they had impairment of effect size 1 or greater for their CR peak timing improvement from block 2 to block 6. In other words, these were participants who were still producing very poorly timed CRs at the end of the conditioning session (classified 'I' in table 5.2), as well as those whose CRs were less poorly timed in block 6 but who had actually deteriorated, having produced better timed responses in block 2 (classified 'T' in table 5.2). One participant was excluded from the timing analysis (D14 in table 5.2 above) because he failed to produce CRs in block 2, however his CRs were well timed in block 6. After the further removal of all participants failing the CR criterion, only one control participant showed impaired timing, compared with ten (77%) of the remaining dyslexic participants. This one control participant also had relatively poor short term memory as assessed by the WAIS-III digit span subtest.

Figure 5.5. Categorisation of participants. *No conditioning* = total CRs <10.8, *Poor tuning* = very inaccurate at end OR slightly inaccurate with little improvement shown.



[13] Effect sizes were calculated as with earlier experiments in this thesis (section 4.3.2.3.).

5.3.4. Secondary analyses

A series of secondary analyses were conducted. It is noted in the method section that some participants reported visual problems, these had no effect on conditioning. An analysis of variance showed a significant effect of gender on the total number of CRs generated, with females producing more conditioned responses [$F(1,30) = 15.80$, $p < 0.001$], but no effect of group and no interaction. A similar 2-between ANOVA revealed only a trend for females to produce greater CR amplitudes [$F(1,30) = 3.36$, $p < 0.1$]. There was no gender effect on block 6 timing error, but a fourth ANOVA revealed a trend for females to show greater improvement in temporal accuracy between the 2nd and 6th blocks [$F(1,17) = 3.53$, $p < 0.1$]. It should be noted that the dyslexic and control groups were well matched for male to female ratio. There were no relationships between conditioning measures and self-reported handedness.

5.3.4.1. Correlational analyses

There were no significant correlations with conditioning measures and age or IQ, furthermore, the dyslexic and control groups were well-matched in these dimensions.

There was a significant correlation (Pearson's $r = 0.38$, $n = 34$, $p < 0.05$) between the maximum response size during calibration and the total number of CRs produced. This reinforces the idea that a greater subjective sense of the US's aversiveness produces stronger conditioning, presumably via an increased motivation to avoid it the US.

Further correlational analyses were carried out to look for relationships between three conditioning outcome measures (Total CRs, CR accuracy at conditioning end and CR accuracy improvement) and 14 psychometric variables (6 WAIS-III subtests, ADD score including and excluding the memory component, Nonsense Word Reading speed and accuracy, WORD spelling and 3 DAST subtests). This produces 42 correlations of which only five were significant at the 5% level. Total CRs produced was negatively correlated with DAST writing fluency ($r = -.376$, $p < 0.05$, $n = 34$). Large timing errors in block 6 were associated with high scores in the block design WAIS-III subtest ($r = .434$, $p < 0.05$, $n = 21$). Large improvements in timing accuracy across the conditioning session were associated with low scores in block design ($r = -.481$, $p < 0.05$, $n = 34$), accurate nonsense word reading ($r = -.507$, $p < 0.05$, $n = 21$) and good WORD spelling scores ($r = .442$, $p < 0.05$, $n = 21$). Two significant results would be expected by chance in 42 tests so these results should be treated with caution.

5.3.5. Debriefing

In response to the questions (Q1 and Q2) addressing awareness, coded response scores were similar for the two groups. On average the dyslexic group scored 2.06 (*s.d. = 0.87*) and the controls scored similarly (2.19, *s.d. 0.75*). When asked if they had been able to predict the next trial's type (Q3), all participants responded

negatively with the exceptions of 5 dyslexics and 3 controls. However, even among those responding positively none claimed to be very successful in their trial type predictions and some reported only considering this for some portion of the session rather than throughout. Only four participants in each group reported that they did not get bored (Q4).

5.4. Discussion

Eyeblink conditioning was used as a direct behavioural test of cerebellar function in dyslexia. A low overall rate of CR acquisition was found in both groups (dyslexic and control). Particularly puzzling were the marginally higher frequency of CR production in the dyslexic group and the failure of several participants to reliably produce CRs. However, the issue of major interest is the dyslexic participants' failure to adaptively time their eyeblinks, as reported by Nicolson *et al.* (2002).

The dyslexic group clearly did not tune their CRs as well as the control group. There is debate in the eyeblink conditioning literature as to the roles of precise locations within the cerebellum during eyeblink conditioning. However the notion that the timing of CRs is a cerebellar cortical process rather than a nuclear task is now a common view (e.g. Garcia & Mauk, (1998); Ohyama & Mauk, (2001)). This notion also fits well with the finding of (predominantly) a timing impairment in the dyslexic samples of the present study and Nicolson *et al.* (2002), as cerebellar influence on language acquisition is surely supplied by the large cerebellar cortical hemispheres, that are unique to human brains, rather than nuclear areas.

The finding of Coffin *et al.*, (2005) that dyslexics fail to acquire classically conditioned eyeblinks was not replicated, in terms of response rates the dyslexic group learned at least as reliably as the control group. A key difference between the present study and the contrasting work is the age of the participants, here adults rather than children (aged ~9 years). The cerebellum retains a high level of plasticity through development and so it is possible that by adulthood it has regained substantial EBC facility despite having none at 9 years of age.

In contrast to the earlier study of adults (Nicolson *et al.*, 2002), there was no evidence of slow habituation to the tone in the dyslexic group compared with the control group. Neither did any dyslexic individual stand out as being unusually slow to habituate.

It should be noted that many participants in this study failed to exhibit the conditioned response with any consistency. Twelve of the 34 failed to reach the conditioning criterion of > 10 CRs in 54 presentations of the conditioned stimulus (48 paired trials). Two of these reported (in response to post-testing interview questions) that they had learned that the beep predicted the air and consequently had ceased blinking because (i) the air was no longer surprising and (ii) it was not sufficiently aversive to avoid. However, the intensity of the US in this study seems to be at least as severe as the typical US in the literature (Daum *et al.*, 1993; Ramnani *et al.*, 2000; Sears *et al.*, 1994; J. A. Tracy *et al.*, 1999). Naturally a major outstanding question is why did so many participants fail to acquire the conditioned response? And if they had acquired it, how well would those responses have been timed?

It was noted in the results section that there was apparently more flexibility in the dyslexic group, in that these participants quickly and dramatically reduced their response rate in the extinction session. This effect may have been a statistical artefact, but if genuine it would fit with previous findings. It closely mirrors the task-switching effect described in the ADT test (section 4.4.2.) and is also paralleled in Nicolson and Fawcett's long-term learning experiments (Nicolson & Fawcett, 2000).

5.4.1. Conclusions

This study has replicated the finding of an impairment in dyslexics' temporal tuning of conditioned responses reported by Nicolson *et al.* (2002). Given the reduced CS-US interval, the results implicate the cerebellum as a cause of abnormal learning in dyslexia. The 38 ms discrepancy between control and dyslexic tuning appears too big to be accounted for by sensory input variability, therefore the results strongly suggest that abnormal cerebellar function, rather than abnormal sensory function, is the cause of the learning differences in this sample. This study again demonstrates the considerable heterogeneity of dyslexia, the cerebellar EBC deficit was not seen in all dyslexic participants, instead, 3 (17%) of the dyslexic participants achieved adaptive conditioning (as indicated by fig. 5.5). Consequently EBC may prove a useful tool for investigation of possible subtypes of dyslexia, and for brain based diagnoses of developmental disorders.

6. Motor skill acquisition

6.1. Introduction

The automatization deficit hypothesis of dyslexia is a cognitive level explanation of the disorder. It proposes that people with dyslexia commonly fail to fully automatize skills they learn, both inside and outside the literacy domain (Nicolson & Fawcett, 1990). Automaticity is a late stage of skill acquisition and could be hampered by difficulties in earlier stages. This study was designed to assess motor skill learning at various pre-automaticity stages across two days to provide a more complete cognitive level description of skill acquisition in dyslexia. A detailed introduction to this study and the relevant literature is given in the introductory chapter.

6.2. Method

6.2.1. Participants

13 Dyslexic and 12 Control participants undertook all aspects of these experiments. The range of ages was 20-24 in both groups, with a mean dyslexic age of 21.46 (s.d. = 1.13) and a mean control age of 22.25 (s.d. = 1.54), (t = 1.47, n.s.). IQ scores (short-form WAIS-III)[14] were also non-significantly higher in the control group (123.38, s.d. 13.43 versus 126.33, s.d. 15.31). Five members of each group were female. Many of these participants were involved in one or both of the previous studies.

6.2.2. Consent and screening

Before the experiments each participant gave informed consent and answered questions probing their baseline levels of common fine motor skills and injury histories. Details on this interview and further questions posed on day two are available in appendix 5. Reports of existing fine motor skill were scored on a 4-point scale. They also completed the Edinburgh Handedness Inventory (Oldfield, 1971) and reported the alcohol they had consumed in the previous 24 hours.

6.2.3. Psychometrics

Many of the participants had volunteered for research previously and consequently much of the psychometric data used was already held by the author. Where testing was necessary this was carried out across the two experimental sessions. As in the balance and eyeblink conditioning studies, the tests undertaken were 6 subtests of the WAIS-III (vocabulary, similarities, digit span, picture completion, digit-symbol

[14] The short-form consisted of the Vocabulary, Similarities, Picture Completion and Block Design subtests.

coding and block design) (Wechsler, 1998), the WORD spelling test (Wechsler, 1993), reading of a passage including nonsense words (Finucci *et al.*, 1976), 3 elements of the DAST (1-minute reading, 1-minute writing, 2 minute spelling) (Fawcett & Nicolson, 1998) and the Brown attention deficit disorder scale (Brown, 1996).

6.2.4. Design

The experiment was designed to monitor participants' learning of a specific short sequence of finger-thumb oppositions. Ability to perform the sequence was assessed twice on day 1 and once on day 2. Training was given between the two assessments on day 1. The testing sessions on day 2 were arranged to be 24 hours after training.

6.2.5. Procedure

The procedure was taken from Korman *et al.* (2003), and both of the sequences used in the former study were adopted by the present study. The sequences were 4-1-3-2-4 and 4-2-3-1-4 with fingers numbered upwards from the forefinger (1). Participants were assigned one of the sequences and instructed to touch the tip of the appropriate finger onto the thumb in the given order. A few repetitions were allowed before the first assessment to ensure that the participant understood the task.

During the training session, participants were asked to perform the 5-contact sequence once every time they heard an auditory tone. The tones were pre-recorded and played back through a computer at a rate of 1 every 2.5 seconds. In total, participants were asked to perform 160 training repetitions over the course of 400 seconds.

Each of the three assessment sessions consisted of 4 30 second blocks in which the participant was asked to repeat the given sequence as many times as possible keeping mistakes to a minimum. Rest periods of 50-60 seconds were allowed between blocks within an assessment session and a 5 minute rest was allowed post training before assessment 2.

6.2.6. Apparatus

Participants wore gloves fitted with copper pads on the thumb and finger tips, which in turn were wired into a computer's USB input via a Personal Measurement Device (http://www.adeptscience.co.uk/products/dataacqu/pmd.html). The computer recorded the start and end of any contact between pads, and the time of each event from the test's start in milliseconds.

6.3. Results

6.3.1. *Psychometric and literacy performance*

Results from psychometric and literacy tests were as expected, with particularly strong between group differences on measures of reading and spelling and the digit-span and digit-symbol coding subtests of the WAIS-III. On average the dyslexic group scored higher on Brown's ADD questionnaire. In fact 4 dyslexics and 2 controls scored above 54 putting them in the "ADD highly probable" range. Key analyses were undertaken with and without these 6 participants.

Table 6.1. Descriptions of the participant groups: Psychometrics, literacy and handedness.

	Dyslexic	Control	t	p
IQ	123.38	126.33	0.51	n.s.
Vocabulary	12.77	14.92	2.58	$p < 0.05$
Similarities	14.15	14.00	0.17	n.s.
Digit Span	9.62	12.42	2.88	$p < 0.01$
Picture Completion	12.62	11.08	1.39	n.s.
Digit-Symbol Coding	9.00	11.50	2.82	$p < 0.01$
Block Design	13.08	13.83	0.63	n.s.
Brown ADD score	54.00	36.33	2.35	$p < 0.05$
NWR Time	79.77	45.42	5.96	$p < 0.01$
NWR Errors	12.23	3.75	6.52	$p < 0.01$
WORD Spelling Score	38.77	46.75	6.89	$p < 0.01$
DAST Reading Score	87.69	116.09	5.12	$p < 0.01$
DAST Writing Score	30.54	35.55	2.39	$p < 0.05$
DAST Spelling Score	29.00	37.55	6.18	$p < 0.01$
Edinburgh Handedness Scale Part 1	0.31	0.61	0.98	n.s.
Edinburgh Handedness Scale Part 2	0.30	0.52	0.88	n.s.
Strength of Hand Dominance	0.70	0.85	1.85	n.s.

Legend: Positive Edinburgh handedness scores denote right-handedness, maximum scores are +1 and -1.

6.3.2. *Skill acquisition data collection*

The experiment consisted of 12 trials of 30 s each, the first 4 were before training and will be referred to as pre-1 to pre-4, the second block of 4 trials were after training on day 1 and will be referred to as post-1 to post-4[15], the final 4 trials followed a delay of 24 hours and are referred to as 24-1 to 24-4. The computer produced an individual file for each trial reporting each contact or end of contact between fingers and thumb, with a different number given for each digit combination. The time of each event was given in milliseconds. A spreadsheet was designed to extract summary information from this raw data.

[15] Or pt-1 to pt-4

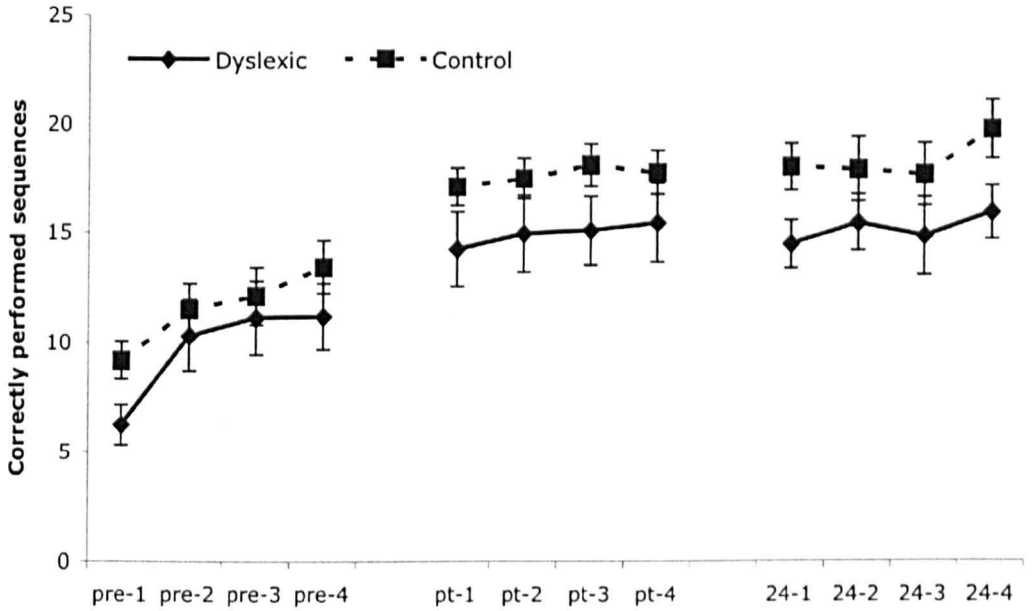
6.3.3. Total correct sequences

A completed sequence was counted when each contact was terminated before the next began and all five contacts were completed in order within the 30 s trial limit.

Inspection of figure 6.1 reveals that the dyslexic group consistently produced less complete sequences in a 30 s trial, this difference was significant at 3 points in the experiment; the very first trial ($t = 2.33$, $p<0.05$), the first trial on day 2 ($t = 2.28$, $p<0.05$) and the final trial on day 2 ($t = 2.10$, $p<0.05$). Removal of participants with high ADD scores leaves the significance of the first test unaffected but reduces the second and third results to trends at 7% and 6% levels of significance respectively.

A 1-between, 1-within ANOVA was performed with GROUP as the between subjects factor and TRIAL as the within subjects factor (2 levels: pre-4 and post-1) to examine performance changes over the training period. There was a highly significant main effect of TRIAL indicating improved performance over training [$F(1,23) = 14.26$, $p<0.01$] but no significant interaction or effect of GROUP. A second ANOVA was carried out in the same way (with within subjects levels of post-4 and 24-1) to examine changes following 24 hours of rest from the task. There was no significant effect of TRIAL and no significant interaction, but there was a trend for the control group to perform more sequences overall [$F(1,23) = 3.10$, $p = 0.09$]. The exclusion of the 6 participants with high ADD scores weakens this trend to non-significance, the pattern of significant results for analyses of variance addressing total number of sequences is otherwise unchanged.

Figure 6.1. Total correct sequences performed



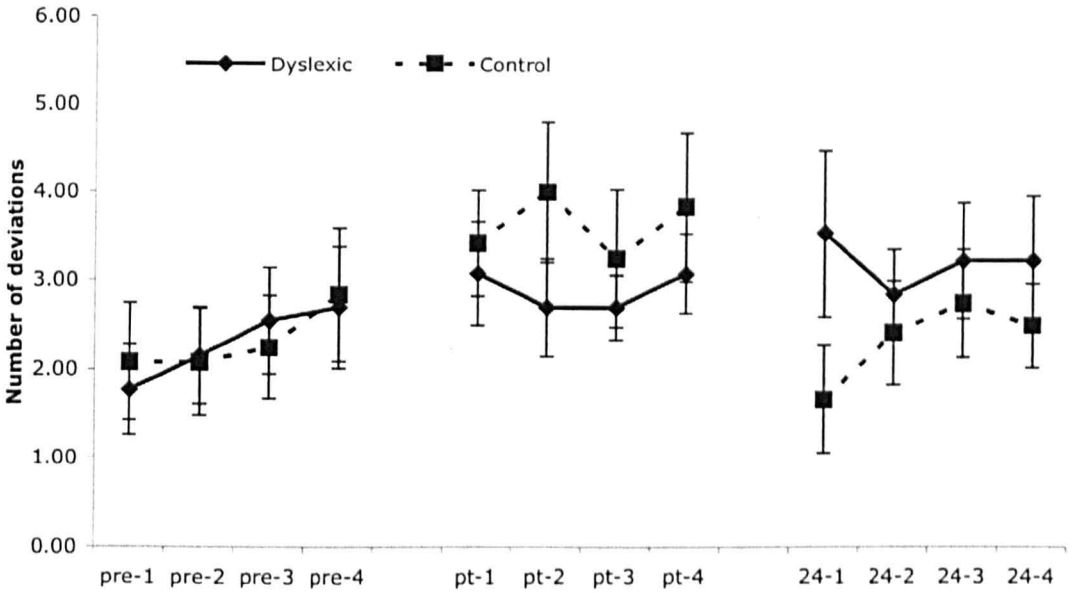
6.3.4. Errors

An error was counted each time the participant performed actions not part of complete correct sequences. Only one error was counted for each deviation from consecutive complete sequences, regardless of how many erroneous contacts were produced during that deviation.

Figure 6.2 shows that numbers of errors recorded for each group were very similar and tended to increase with the increasing speed of performance through the pre-training block. Later in the experiment there is an apparent dissociation whereby control subjects produced most errors immediately after training and dyslexic participants were most error prone on day 2. However, within group variance is high and consequently there is no significant between group difference in any trial.

Analyses of variance were performed for error scores analogous to those used for complete sequences in order to examine changes in performance with training and overnight. There were no significant effects in either analysis, but there was a trend towards an interaction, whereby controls eliminated errors between post-4 and 24-1 with no such error reduction in the dyslexic group [$F(1,23) = 3.66$, $p = 0.07$]. Removal of participants with high ADD scores strengthened this effect [$F(1,17) = 6.27$, $p < 0.05$] but leaves all other results non-significant.

Figure 6.2. Number of errors made in each trial.

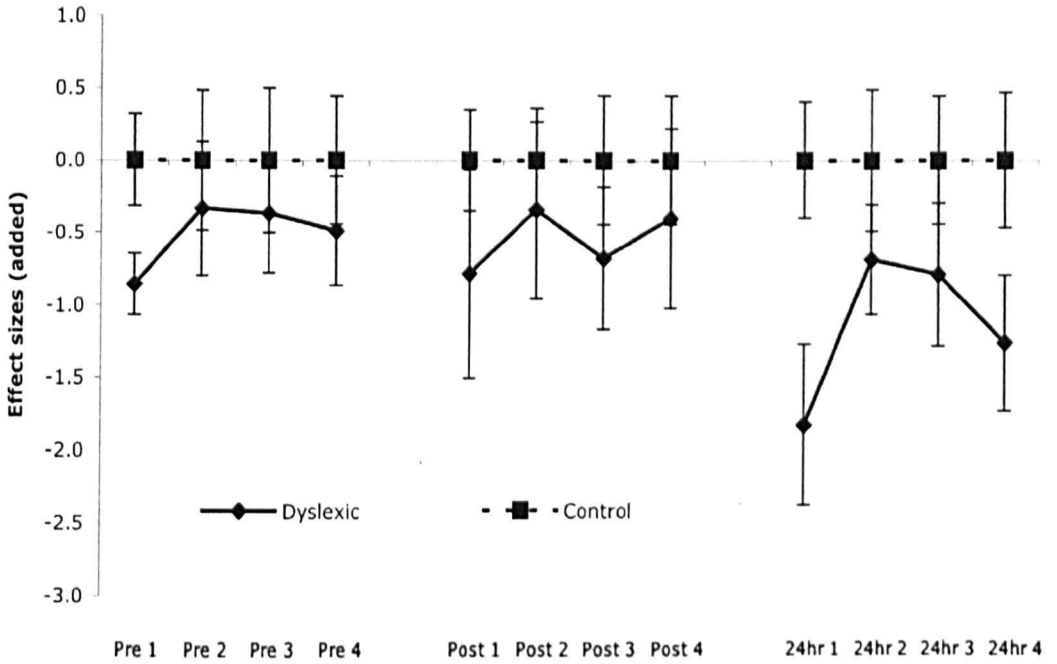


6.3.5. Combination of speed and accuracy

Interpretation of performance speed and error data is complicated by the fact that the two are in conflict. In order to obtain a combined speed and error measure, to reflect overall competence, individual effect sizes were calculated (see section 4.3.2.3.). Effect sizes for errors and total complete sequences were summed for each trial. Hence, figure 6.3 shows control performance consistently at zero by definition, with standard error bars describing within group variance. Dyslexic group scores indicate the mean sums of participants' deviations from the control group means in standard deviation units, for the two parameters of speed and accuracy (equally weighted).

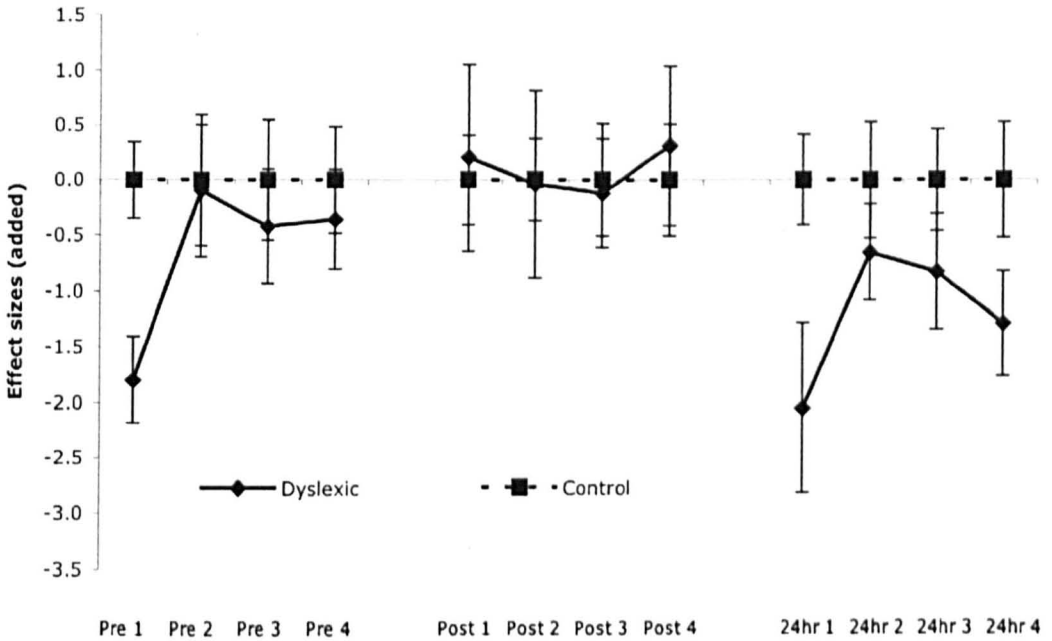
The combined speed and accuracy scores reinforce the emerging picture of generally impaired performance in the dyslexic group, with particular deficits at the beginning of the experiment, at return on day 2 and in the experiment's final trial. 2-tailed independent groups t-tests revealed significant differences for the first two of these ($t = 2.27$, $p < 0.05$; $t = 2.62$, $p < 0.05$; $t = 1.90$, $p = 0.07$, respectively).

Figure 6.3. Combination of total correct sequences and errors.



The picture becomes yet clearer with the removal of the participants with high ADD scores, as shown in figure 6.4, with near identical performance of the two groups seen in the post-training session. The pattern of significant results from t-tests was unchanged ($t = 3.48$, $p < 0.01$; $t = 2.44$, $p < 0.05$; $t = 1.83$, $p = 0.08$, respectively).

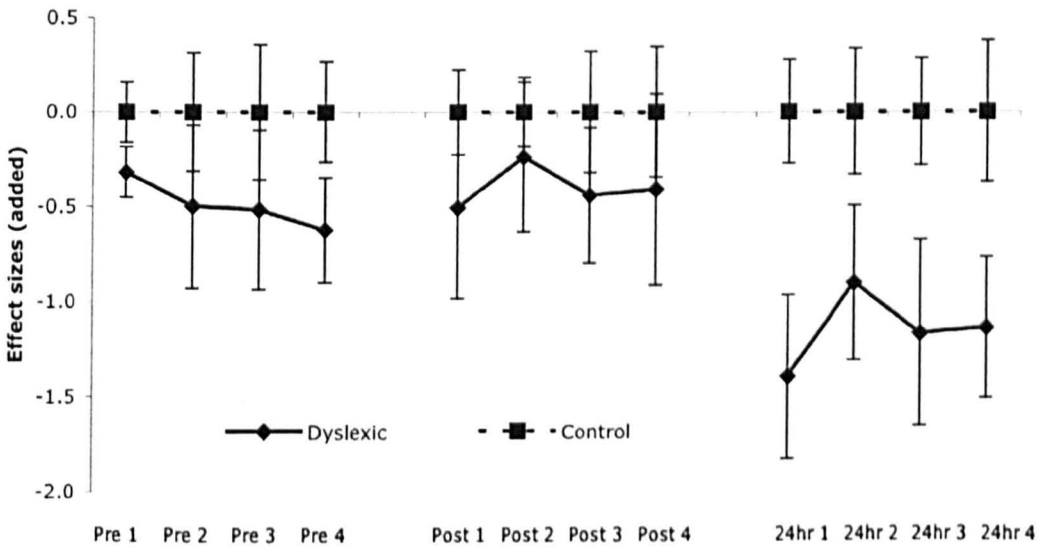
Figure 6.4. Combination of total correct sequences and errors excluding data from participants with high ADD scores.



One concern with this analysis is that it could be argued that the measures of speed and accuracy are not directly opposed. Since the speed component is actually 'total correctly completed sequences', this could be similarly low for a participant who had produced fast movements and consequently a lot of errors, or someone who had exercised a moderate pace but made few errors. To check the results above were not the product of this issue, a second analysis was performed replacing the 'total correctly completed sequences' count with a measure of raw speed, a simple count of the number of finger-thumb contacts made in the trial (regardless of their appropriateness).

A similar pattern was obtained (see figure 6.5), with pronounced dyslexic deficit immediately after the 24hr break. In contrast to the results presented above however, there appears to be similar performance in each group at the start of the experiment. Between group differences are significant or near significance at trial 24hr-1 (t = 2.69, $p < 0.05$), trial 24hr-3 (t = 2.02, $p = 0.055$) and trial 24hr-4 (t = 2.17, $p < 0.05$).

Figure 6.5. Combination of raw performance speed and number of errors.



6.3.6. Correlations

Correlational analyses were carried out to investigate relationships between psychometric indicators of dyslexia and the combined (total correct sequences and errors) measures of the motor skill acquisition (MSA) task. Several significant relationships were found. Combined performance in pre-1 was negatively related to NWR accuracy, such that those who made most reading errors were most impaired in the MSA experiments' first trial ($r = -0.420$, $p<0.05$). This relationship was also evident in trial 24-4 ($r = -0.444$, $p<0.05$), but at this final stage there was an even stronger correlation with digit span, poor working memory being associated with poor MSA ($r = 0.655$, $p<0.001$). However, the most comprehensive link between literacy and MSA was seen immediately after the 24hr break, with poor MSA performance associated with slow NWR ($r = -0.513$, $p < 0.01$), inaccurate NWR ($r = -0.429$, $p<0.05$), poor WORD spelling ($r = 0.547$, $p<0.01$) and poor DAST 2-minute spelling ($r = 0.559$, $p<0.01$).

A further combined (speed and accuracy) measure 'improvement' was constructed[16] to reflect the change from post-4 to 24hr-1. Those showing least improvement tended to read the NWR passage slowly (r -0.552, $p<0.01$).

None of these four measures of MSA performance were correlated with alcohol consumption, handedness or hours of sleep between testing sessions.

6.3.7. Spearman non-linear correlations

A Spearman correlational analysis was undertaken because scatter plots indicated that Pearson's test may have been excessively influenced by outliers. This second analysis showed that good performance at pre-1 was associated with accurate NWR performance ($r = -0.451$, $p<0.05$), this relationship was also found at 24hr-4 ($r = -0.445$, $p<0.05$) and immediately after the overnight break ($r = -0.485$, $p<0.05$). Also following the overnight break (at 24hr-1) performance was significantly related to digit-span ($r = 0.428$, ($p<0.05$), WORD spelling ($r = 0.529$, $p < 0.01$) and DAST spelling (0.501, $p<0.05$). The overnight improvement measure was related only to DAST writing ($r = 0.407$, $p<0.05$). It was surprising to note that MSA performance at the end of the experiment (24hr-4) was negatively related to hours slept between test days ($r = -0.473$, $p<0.05$) since effective consolidation is supposedly dependent on sleep (Walker *et al.*, 2002). Hence, performance in this post sleep stage (24hr-4) could have been expected to be best when participants had slept for longer, especially as stage 2 NREM sleep late in the night was found by Walker and colleagues to be most strongly related to improved performance.

6.3.8. Individual analyses and participant categorisation

Dyslexia appears to be related to performance of this MSA task at 3 points, pre-1, 24hr-1 and 24hr-4. Hence, individual analyses were conducted to see how well combined speed and error performance at each of these three points separated dyslexic and control participants. An individual was said to be impaired on any of these 3 variables if they produced a performance at least 1 effect size below the control mean performance when speed and error effect sizes had been summed to account for the speed-accuracy trade-off.

[16] The change in total correct sequences and the change in simple errors were calculated and the effect sizes for these measures were then summed to produce "improvement".

Figure 6.6. Prevalence of MSA impairments

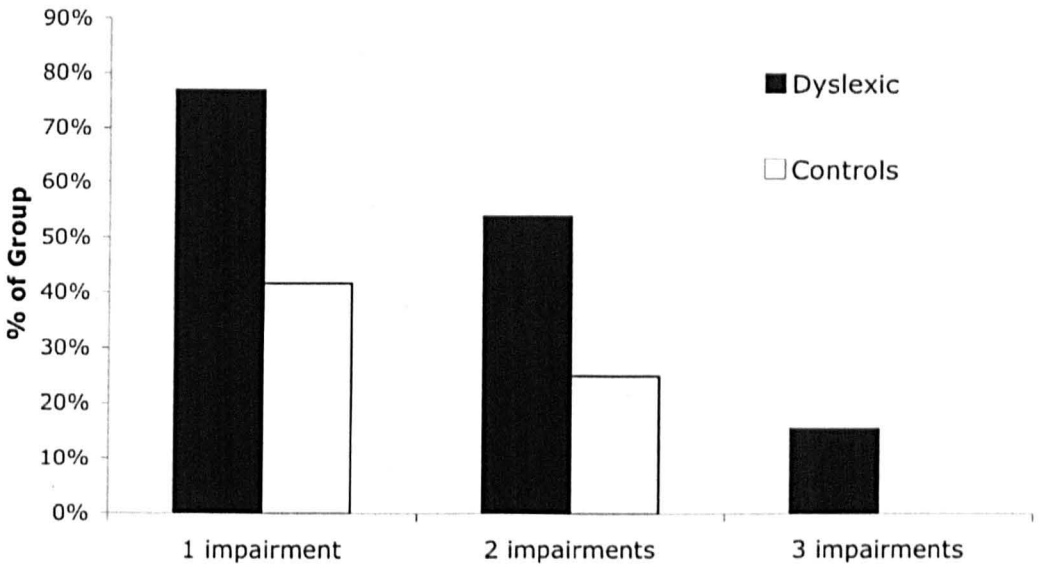


Figure 6.6 shows that 77% of the dyslexic group showed MSA impairment on 1 or more of the 3 key stages, compared with only 42% of the control group. The ratio is preserved at around 2:1 for 2 impairments. No control participant was impaired at all three points.

On the 'improvement' measure (combined speed and errors) there was impairment (-1 effect size or worse) in 46% (6/13) of the dyslexic group and 25% (3/12) of the control group. Therefore, as an overall prevalence rate of MSA impairment in dyslexia (particularly in consolidation), 46-77% seems appropriate.

Participants' self reports of existing motor skills were scored on a 4 point scale, with higher scores representing higher levels of expertise. An independent groups t-test confirmed that there was no difference between groups ($t = 1.06$, n.s.).

6.4. MSA discussion

In line with previous research (e.g. Nicolson & Fawcett, (2000)) there was a general trend of enhanced motor performance in the control group relative to the dyslexic group but the learning rates of the two groups appeared to be equivalent during explicit practice. There is evidence of a specific deficit in consolidation for the dyslexic group, shown as a particular performance deficit immediately after the overnight break. This deficit had long lasting implications, the between groups difference was still significant by the last trial of the experiment when the dyslexic group had had time to catch up through conscious effort, as they appeared to do in the first session (before training on day 1). These results parallel findings reported by Shea *et al.* (2000) on the spacing of practice sessions. The dyslexics' ability to catch up from initial deficit during the pre-training session in the present study is itself supportive of the automatization deficit hypothesis of dyslexia, which proposes that persons with dyslexia often achieve normal skill performance levels by "consciously compensating" (Nicolson & Fawcett, 1990). The consolidation impairment seen in dyslexics in this study could be expected to contribute automatization failure, since the skill consolidation stage precedes automaticity in normal motor learning.

6.4.1. Co-morbidity

The present sample included 6 participants with high ADD scores on the Brown scale (Brown, 1996). Since co-morbidity is such a prominent topic in current dyslexia research they were not excluded entirely, but instead the results were examined with and without these individuals. The key difference seen with the removal of ADD participants was much closer performance of the dyslexic and control groups immediately after training. This could easily have been expected, it seems likely that participants with poor attention would gain least from prolonged explicit practice of a simple motor sequence. The near identical performance of 'pure' dyslexics and controls post-training is a strong indication that the consolidation effect is not due to a general failure to learn skilled behaviours or to a general lack of attention in the dyslexic group. These results additionally indicate that it is those people who have overt symptoms for more than one of these supposedly discrete disorders who suffer most in terms of performance of a target behaviour.

6.4.2. Heterogeneity

Along with co-morbidity, heterogeneity in dyslexia is a theme whose importance is being increasingly acknowledged. The case for careful attention to heterogeneity is further strengthened by this study. While all the participants met the diagnostic criteria for dyslexia, not all showed the clear consolidation deficits that some exhibited. This does not undermine the importance of the high prevalence of consolidation deficit seen in the dyslexic group, but opens exciting possibilities for

specialised remediation programs and treatments for various subtypes of dyslexia as they emerge.

6.4.3. Underlying brain circuitry

This is certainly not a straight forward issue, but one that lends itself to examination from numerous angles. Firstly it should be noted that while Ohno *et al.* (2002) observed that deprivation of REM sleep seemed to suppress cerebellar learning in classical eyeblink conditioning, it was stage 2 NREM sleep that was most strongly associated with effective consolidation in MSA experiments conducted in another lab (Walker *et al.*, 2002), implying a dissimilar involved neural process or processes.

As mentioned in the introduction, Doyon and Benali (2005) distinguish between two types of motor skill acquisition, motor adaptation and motor sequence learning. The task used by this study falls in the latter category. According to their model, early acquisition of either type involves both cortico-striatal and cortico-cerebellar systems. However, once a skill is well learned its neural representation becomes confined to one system or the other, with the cortico-striatal system relevant to motor sequence learning and the cortico-cerebellar system crucial for long term memory of motor adaptation learning.

Following this distinction it is hard to draw strong conclusions relating the present results to biology. Initially because of the vagueness of the phrase "well learned", participants in the present study were required to complete 160 trials in the training session alone, but given the importance of distribution of practice across days for optimal skill acquisition (Shea *et al.*, 2000) it is not clear whether such intense practice could be considered to produce a "well learned" motor sequence. Whether or not performance here was well learned, (and consequently whether the cortico-cerebellar and cortico-striatal systems or solely the latter system were involved at the end of the task) there are still several brain structures involved. Furthermore, according to the model both systems were involved at the start of the experiment and so any deficits in late performance could still be attributable to ineffective processing in the cortico-cerebellar system in the early stages of learning. Expert performance of the sequence is too fast to be governed by a feedback system. The cerebellum's role in predictive timing suggests involvement in the development of smooth feed-forward motor programs, this is consistent with the proposition that impaired performance at a late stage in the learning of this task could be the result of earlier abnormal function in the cortico-cerebellar system.

Of the two most prominent biologically founded theories of dyslexia, the MDH is the less difficult to apply here. Since the test trials did not include any auditory feedback or cues, and visual feedback was obstructed, the theory makes no strong predictions. The proposition of impaired pan-sensory timing might relate to tactile feedback from fingers and thumbs, but the timing deficits proposed by the Magnocellular hypothesis are too small to be expected to mix the order of input, with gaps between contacts in performed sequences typically in the region of 163

ms. Furthermore, any disruption of tactile feedback might be expected to impair performance in the early stages of learning rather than in the more automatic stage post-consolidation, since feed-forward strategies are likely to be employed at the later stage. This reinforces the point that impaired sensory input is not likely to be the cause of impaired performance on day 2.

Regarding the CDH, the picture is a little more complicated. The Doyon and Benali (2005) framework would expect cerebellar involvement in the initial learning of the motor sequences, but we have seen it is hard to pin down exactly how early its involvement is supposed to end. So perhaps abnormal cerebellar function is behind the poor acquisition of the dyslexic group in trial 1, or if the appropriate time frame is in fact the whole first day then there is little case for abnormal cerebellar function, but again, if normal cerebellar function is important for optimal consolidation over the first 24 hours, then abnormal cerebellar function could be the cause of the impaired consolidation exhibited by some dyslexic participants.

Traditional views of the role of the cerebellum in motor skill place emphasis on timing (Ivry, 2000; Ivry & Keele, 1989; Sakai *et al.*, 2002) and on error-correction following feedback (Doya, 2000). The task used in this study had no specific timing requirement, however, timing deficits have previously been reported in dyslexia (Nicolson *et al.*, 1995). There was also no explicit feedback on accuracy in this task, which may have precluded strong cerebellar involvement, with the cerebellum thought to be particularly involved in "supervised learning" (Doya, 2000). A PET study showed abnormal cerebellar activation in dyslexia when a simple motor learning task did include explicit feedback on accuracy and performance was required at a fixed pace (Nicolson *et al.*, 1999). Classical eyeblink conditioning provides an example of simple supervised learning mediated by the cerebellum (for a review of the biology of eyeblink condition see Hesslow & Yeo, (2002)). In eyeblink conditioning paradigms, inter-trial intervals of around 20s are typical for human adults and it is thought that considerably shorter intervals could be detrimental to conditioning, perhaps activation might be confused from one trial to the next. It may be that the minimal time gaps between repetitions (normally < 1 s) in the present study were an obstacle to optimal supervised cerebellar learning.

Finally on the subject of MSA and the cerebellar deficit hypothesis, I should address the issue of lateralization. In this study participants were tested on their non-dominant hand, therefore in most cases they used their left hand. Presumably then, the left rather than right cerebellum would be involved primarily. However, one explanation of the heterogeneity of dyslexia's manifestations is that there might be a variety of location of cerebellar abnormality: Although all dyslexics' literacy impairments could theoretically be caused by cerebellar dysfunction, other parts of the cerebellum may be left unaffected, with only the specific affected parts producing signs of cerebellar deficit. The cerebellar hemispheres are connected to contralateral cerebral cortex and therefore, right-handed participants using their left hand for the MSA task were engaging the side of the cerebellum that is not extensively involved with cerebral language areas, and by implication, the side of

the cerebellum that could easily be functioning normally in a person with extreme reading and spelling difficulties that are caused by cerebellar deficit. Regardless of the issue of cerebellar involvement, one might expect stronger between group performance differences had the dominant hand been used, since there was a trend for persons with dyslexia to have a weaker preference for their dominant hand as assessed by the Edinburgh handedness inventory ($t = 1.85$, $p = 0.08$) (Oldfield, 1971).

6.4.4. Conclusions

A dissociation was observed whereby dyslexic participants seemed able to learn the sequence at a comparable rate to the controls within an initial session of explicit tests. However, the same participants fell behind after a night's sleep. This suggests an abnormality of consolidation processes. This detriment was sufficiently severe that a final session of explicit tests was not enough practice for the dyslexic group to again match the controls. It is possible, therefore, that the documented automatization deficit in dyslexia may be rooted further back in the learning process.

7. Cross-study comparisons and discussion

7.1. Introduction

Many studies of motor skill and skill acquisition outside the literacy domain have shown impairments in dyslexia at the group level, however, investigations that have probed deeper and examined individual participant data have found that such deficits are rarely (if ever) universal in a sample diagnosed with dyslexia. For example, Fawcett and Nicolson (1999) reported one of the highest rates of motor impairment after administering a battery of clinical tests for cerebellar dysfunction. They found evidence of abnormality in muscle tone or postural stability in over 95% (but not 100%) of their dyslexic participants. In a more recent study, Moe-Nilssen *et al.* (2003) reported that parameters of walking performance could correctly classify over 77% of their dyslexic and control participants to one of those groups. Franck Ramus and colleagues (Ramus *et al.*, 2003b) tested dyslexic students on a range of tests of phonological, visual, auditory and cerebellar processing tests and reported that all had phonological problems, with 2/16, 8/16 and 4/16 showing visual, auditory and cerebellar problems respectively. The three studies reported in this thesis have produced comparable findings, with overall impairment prevalence rates in dual-task balancing, eyeblink conditioning and motor skill acquisition of ~53%, ~77% and ~46% respectively, depending on the precise criterion chosen (as opposed to 15%, 13% and 25% in the control group).

These individual effect size analyses are important because studies of non-literacy deficits are often designed to throw light on the underlying biological cause(s) of dyslexia. Strong impairments in some but not all dyslexic participants point towards the possibility of multiple causations of what is traditionally considered a single discrete disorder. Multiple separate causes would surely mean multiple disorders and therefore multiple, specific remediation and screening programs.

Data from key measures in all three studies were available from fourteen participants (8 dyslexic, 6 control) who undertook all three sets of experiments and so it is possible with individual effect size analyses to compare the performances of some participants across studies.

7.1.1. Commonalities and contrasts between the three studies.

All three of the studies in this thesis examined the differences between dyslexic and control participants in their motor performance and or learning. All three studies found significant between group effects on the key motor variables together with considerable within group variability, especially for the experimental group. Closer inspection of the data revealed that between group differences tended to reflect substantial deficits for some but not all of the participants involved.

In terms of biology, the cerebellum is well-known to be a key structure in motor co-ordination and motor skill acquisition (Thach, 1998). Clearly it will have been involved to some extent in all of the experimental tasks. Briefly and more specifically, the cerebellum is strongly implicated in balance (Ouchi *et al.*, 1999), and the specific balance posture used in the present experiments (heel-to-toe) seems to be particularly dependent on cross-hemispheric communication in the cerebellum (Bastian *et al.*, 1998). Classical conditioning of eyeblinks was studied expressly for the reason that it is probably the best single behavioural test of cerebellar function, since the involved neural circuits are well researched and characterized, and are centred on the cerebellum (Hesslow & Yeo, 2002). The rationale for the motor skill acquisition study was more at the cognitive level than the biological, primarily aimed at probing the consolidation learning stage. While the cerebellum is undoubtedly active in the acquisition of this skill, cerebral cortical areas and the basal ganglia are likely to be more important (Doyon & Benali, 2005).

7.1.2. Rationale for cross-study analysis

Despite the commonalities between the three empirical studies as outlined above, it is clear that they are also rather diverse. It would therefore be striking to find any pattern of deficits across the studies and it is important to make use of this opportunity to examine whether such a pattern exists. The emergence of consistent patterns of deficits across the studies could inform the development of understanding of subgroups in dyslexia. For example, if it were to emerge that some participants had deficits in error elimination during sequence learning and in EBC, it may be said that there exists a subgroup of dyslexia primarily characterized by a general deficit in error reduction across learning modes. Alternatively if balance and EBC deficits commonly co-occur in the absence of MSA difficulty there may be abnormality of cerebellar processing without impairment of basal ganglia or cerebral cortex. Furthermore, Nicolson *et al.* have suggested that the variety of observed impairments in persons with dyslexia could point to a variety of location of cerebellar abnormality (Nicolson *et al.*, 2001).

7.1.3. Predictions

It is predicted that dyslexic participants showing clear impairment in the experimental aspects of one study will be more likely to show impairments in one or more of the other studies. Conversely, it is hypothesised that control participants' deficits in balance, EBC or MSA are spurious and will not be seen in other domains.

7.2. Method and results

Effect sizes were calculated (see section 4.3.2.3) for all participants in all studies for the dyslexia diagnostic variables as well as for key experimental variables from each study. Signs were arranged so that positive scores always represented 'better' performance than the control mean. Participants were selected for these analyses if data were available from them for all three studies. Two participants who took part in all studies were not included in this analysis because their balance data was missing due to technical failure. It should also be noted that participants C1, C2 and C4 failed the conditioning criterion and consequently CR timing data were not available rendering these participants unable to show timing impairment. Further, note that the participant identifiers "C1", "D3" etc. (employed in the following tables and figures) do not necessarily refer to the same individuals that they referred to in previous chapters.

Two participants in this chapter had produced ADD scores in the "highly probable" range. These were D4 and C2.

The following tables show the relevant effect sizes for all the remaining participants (8 dyslexic and 6 control), split into psychometric, balance, EBC and MSA for clarity.[17]

[17] The effect sizes are derived from the EBC control group for the psychometrics and EBC, but from the other respective control groups for the other experimental tests.

Table 7.1. Psychometric profiles (effect sizes)

D. ID	D1	D2	D3	D4	D5	D6	D7	D8	Mean
Vocab.	-1.75	-0.57	-1.36	-1.75	-0.57	-1.36	-1.36	-0.96	-1.21
Similar.	-0.97	0.71	0.29	1.13	1.96	1.13	-0.13	2.38	0.81
Digit Span	-1.43	-1.43	-0.51	-0.97	1.31	-2.34	-2.34	-0.97	-1.09
Picture Comp.	-0.45	1.35	0.45	0.75	0.75	0.45	0.45	0.75	0.56
D-S Coding	-0.91	-0.55	-1.28	-0.91	-0.91	-1.28	-0.55	-1.28	-0.96
Block Design	1.31	0.49	-0.75	0.90	1.31	-1.16	-2.81	-0.33	-0.13
NWR Speed	-1.26	-3.79	-1.64	-3.98	-4.73	-3.61	-0.98	-1.83	-2.73
NWR Acc.	-2.24	-5.64	-1.76	-5.15	-2.73	-3.21	-6.12	-2.24	-3.64
WORD Spelling	-2.57	-6.57	0.29	-6.00	-4.28	-3.71	-4.28	-3.14	-3.78
DAST Reading	-2.03	-2.54	-1.01	-3.79	-0.64	-2.40	-0.57	-1.59	-1.82
DAST Spelling	-1.81	-2.47	-3.13	-2.80	-3.13	-1.47	-2.47	-1.47	-2.35
DAST Writing	-1.56	-1.84	-4.65	-1.56	-1.84	-0.72	-0.44	-1.28	-1.74
ADD Scale	-0.16	-1.01	-0.95	-3.77	-0.89	-0.34	-0.59	1.01	-0.84

C. ID	C1	C2	C3	C4	C5	C6	Mean
Vocab.	-0.17	1.01	0.62	-0.57	0.62	-1.36	0.02
Similar.	0.71	-0.55	-0.13	0.29	1.13	-0.97	0.08
Digit Span	0.40	-0.51	-0.06	-0.06	2.23	-1.43	0.10
Picture Comp.	0.45	-0.75	-1.05	-0.45	1.35	-0.75	-0.20
D-S Coding	0.18	0.91	-1.65	-0.18	1.65	-1.28	-0.06
Block Design	0.90	-0.75	-1.57	-0.75	0.90	-0.75	-0.33
NWR Speed	-0.23	0.33	-0.05	0.42	1.08	0.52	0.34
NWR Acc.	-0.79	-1.27	1.64	0.18	1.64	0.18	0.26
WORD Spelling	0.29	-0.29	-0.86	0.86	0.29	1.43	0.29
DAST Reading	1.19	0.53	-1.67	0.16	0.60	0.16	0.16
DAST Spelling	1.18	-0.48	-1.81	0.19	-0.15	1.18	0.02
DAST Writing	-0.44	1.25	-0.72	-0.72	-0.44	-0.72	-0.30
ADD Scale	-1.01	-1.26	-0.10	1.31	0.27	1.37	0.10

Table 7.2. Heel-to-toe balance performance profiles (effect sizes).

D. ID	D1	D2	D3	D4	D5	D6	D7	D8	Mean
Balance alone	0.40	-6.92	-0.19	1.02	-0.94	0.48	-0.83	-2.36	-1.17
Balance blindfold	0.79	-1.85	-0.76	1.05	-0.34	0.25	0.04	-3.71	-0.57
Balance while count-ing	-5.78	-2.35	•	-3.44	-0.18	0.79	-3.86	-4.70	-2.79
Balance with slow CRT	-7.83	-1.15	-0.36	-0.37	-0.94	1.08	-6.44	-3.67	-2.46
Balance with fast CRT	-2.09	-1.40	0.02	-1.22	-0.27	1.01	0.43	-4.66	-1.02

C. ID	C1	C2	C3	C4	C5	C6	Mean
Balance alone	-0.11	-1.28	0.94	0.15	-0.52	0.44	-0.06
Balance blindfold	0.04	-1.08	1.13	0.41	-0.43	0.76	0.14
Balance while coun-ting	0.24	-1.87	1.04	0.20	-0.69	-2.19	-0.55
Balance with slow CRT	-0.34	-2.54	0.96	0.30	-0.70	0.11	-0.37
Balance with fast CRT	-0.75	-2.60	0.70	-0.52	-0.20	0.18	-0.53

Table 7.3. Eyeblink conditioning profiles (effect sizes)

D. ID	D1	D2	D3	D4	D5	D6	D7	D8	Mean
Total CRs in Conditioning Session	1.69	0.59	1.81	1.20	-0.21	0.77	0.96	0.71	0.94
Timing error in block 6	-1.31	-0.24	-2.26	-0.50	0.79	0.14	1.29	-2.86	-0.62
Reduction in timing error from block 2 to block 6	1.34	-2.16	-0.56	0.19	-0.99	-1.82	0.57	-2.10	-0.69
C. ID	C1	C2	C3	C4	C5	C6			Mean
Total CRs in Conditioning Session	-0.88	-0.82	0.83	-0.88	1.26	0.83			0.06
Timing error in block 6	•	•	1.17	•	0.78	0.57			0.84
Reduction in timing error from block 2 to block 6	•	•	1.18	•	-0.01	-0.15			0.34

Table 7.4. Motor skill acquisition profiles (effect sizes, all based on the raw speed-simple error count combination measure)

D. ID	D1	D2	D3	D4	D5	D6	D7	D8	Mean
Pre-1	-0.18	-0.78	0.29	0.30	-0.60	-1.10	-0.40	-0.82	-0.41
24-1	-0.36	-2.22	-1.09	-0.55	0.32	-0.76	-2.20	-0.94	-0.98
24-4	-0.06	-1.93	1.15	-0.15	-0.08	-1.24	-2.72	-0.89	-0.74
C. ID	C1	C2	C3	C4	C5	C6			Mean
Pre-1	-0.38	0.38	0.65	0.71	-0.70	-0.52			0.02
24-1	1.63	-1.01	0.21	1.21	-0.06	-1.27			0.12
24-4	0.69	0.37	-0.10	-0.15	2.47	-2.68			0.10

The next stage in this analysis was to categorize each of these 14 participants as "impaired" or "unimpaired" for each of these variables, taking a cut off point of -1 effect size or worse as indication of impairment. That is, performance at least 1 control standard deviation worse than the mean control level. The following graphs show the percentage of each group impaired on various psychometric and experimental variables.

Figure 7.1. Prevalence of impairment on psychometric and literacy variables

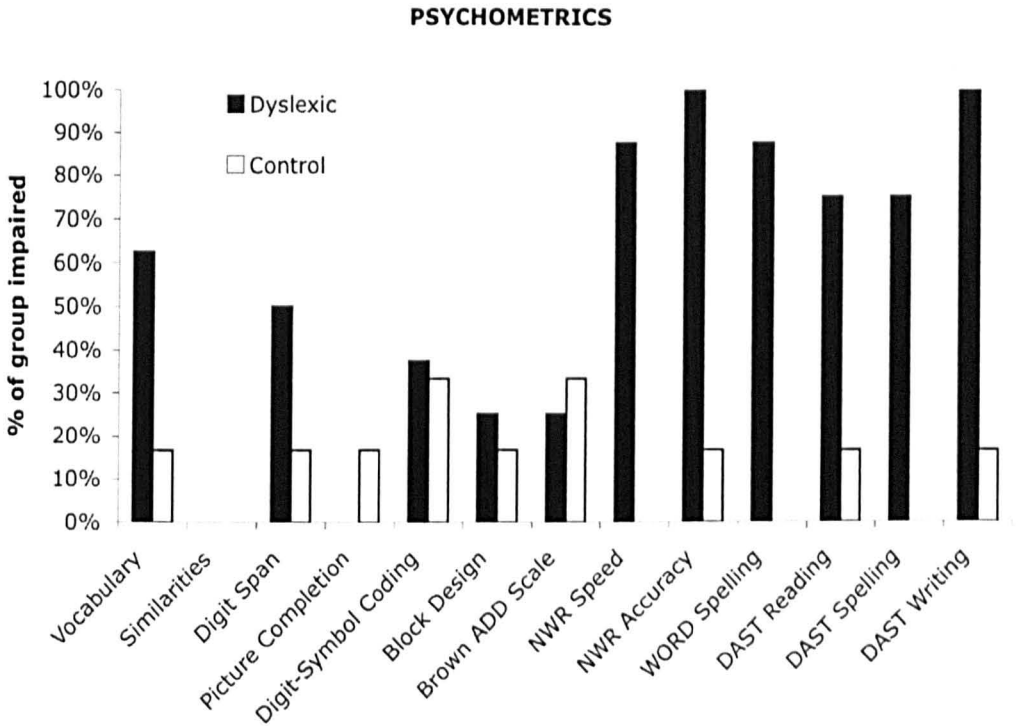
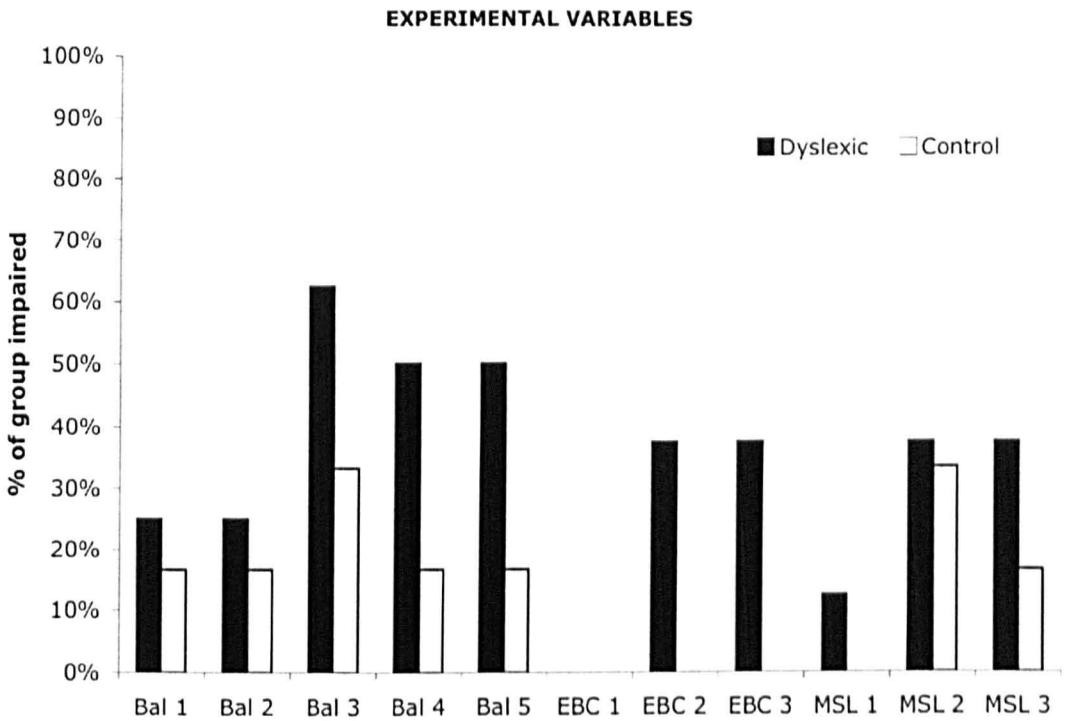


Figure 7.2. Prevalence of impairment on experimental variables

Legend: Bal 1 = Balance alone, Bal 2 = Balance blindfolded, Bal 3 = Balancing while counting, Bal 4 = Balancing during slow CRT, Bal 5 = Balancing during fast CRT, EBC 1 = Total CRs in conditioning session, EBC 2 = Timing error in block 6, EBC 3 = Timing improvement from block 2 to block 6, MSA 1 = Performance in pre-training trial 1, MSA 2 = Performance in day 2 trial 1, MSA 3 = Performance in day 2 trial 4.



As expected, dyslexic impairments are most consistent for the diagnostic reading and spelling variables. However, dual-task balance tasks and EBC timing also cause impairment much more frequently in the dyslexic group than in the control group.

7.2.1. General comparison across studies within participants.

In this analysis, each of the areas of assessment was reduced to a single impaired/unimpaired decision.

Psychometrics – Eight variables were taken as indicators of dyslexia (Digit-Symbol Coding, Digit Span, NWR speed, NWR accuracy, WORD Spelling, DAST reading, DAST spelling, DAST writing), participants were considered impaired if they showed effects sizes of -1 or worse on 4 or more of these 8 measures.

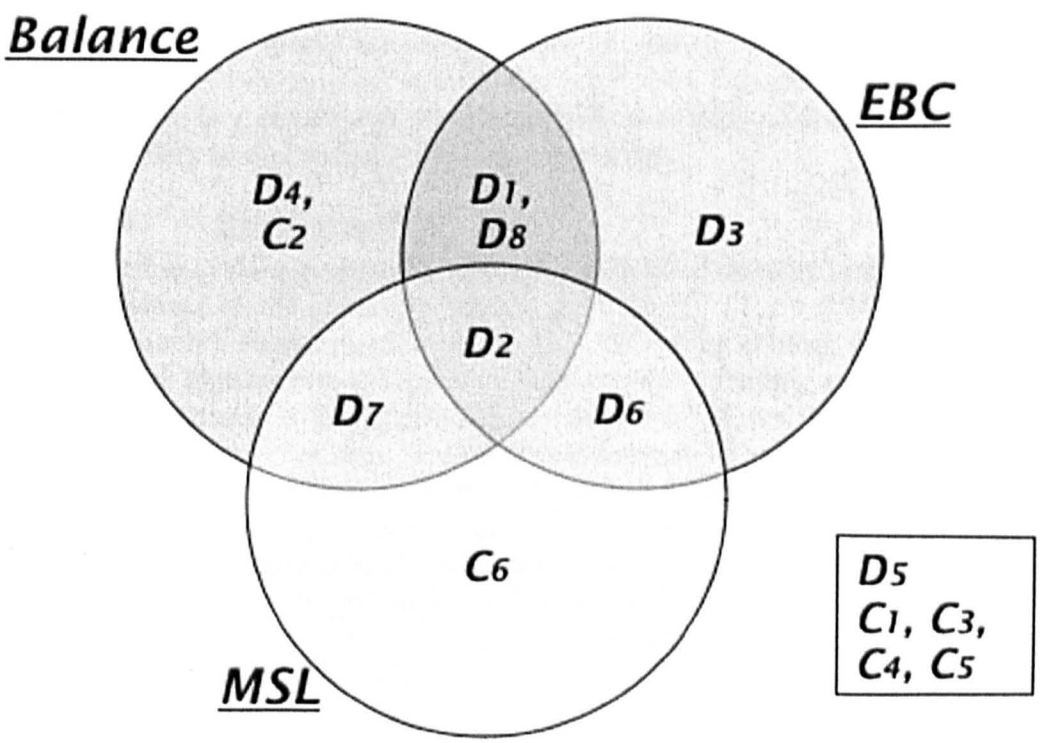
Balance – This analysis considered overall impairment in the balance study where participants produced effect sizes of -1 or worse on 2 or more of the 3 tests of dual-task balance.

EBC – Participants were considered to have deficits in adaptive eyeblink conditioning if they showed an effect size of -1 or worse for either their CR timing improvement or their final CR accuracy.

MSA – MSA impairment was recorded when participants produced 2 or more effect sizes of -1 for their performances at 3 points in the experiment (pre-1, 24-1, 24-4). The measure used was the simple error-raw speed combination.

Using these criteria, all 8 dyslexic participants showed overall impairment on psychometric dyslexia indicators, compared with none of the controls. For balance the figures were 5/8 to 1/6, for EBC timing 5/8 to 0/6 and for MSA 3 of 8 dyslexic participants showed overall impairment to 1 of 6 control participants. Taking a step further, no control participant was impaired in more than 1 study using this analysis, 3 dyslexics were impaired in balance and EBC, 2 in balance and MSA and 2 in EBC and MSA (including 1 who showed impairment in all 3 studies). These overlaps are demonstrated in the following diagram.

Figure 7.3. – Venn diagram showing distribution of participants' performances across studies.



Clearly there is not any pattern of great strength, however, two points are worth making.

1) Neither of the control participants showing deficit in experimental variables here had difficulty in more than one area. Therefore, if these impairments in balance, EBC and MSA are symptoms of a common underlying cause (which may also underlie dyslexia), it is quite plausible that that cause is not present in those two controls, but that their impairments are due to some other variable.

2) Of the 7 dyslexic participants showing impairment in experimental areas, 5 had balance problems and 5 had EBC timing problems, whereas only 3 had difficulty with MSA. All of those 3 also had difficulty with balance or EBC timing or both. This suggests that the first 2 studies are more strongly tapping the systems that do not function properly in dyslexia. Specifically, it seems that the MSA task relies less heavily on the cerebellum. For example a very recent imaging study (Walker *et al.*, 2005) employing a virtually identical task concluded that the cerebellum is one of several structures involved in sleep-dependent consolidation, whereas functional normality of the cerebellum is crucial for normal eyeblink conditioning (Hesslow & Yeo, 2002) and selective posterior vermal lesions of the cerebellum have severe effects on heel-to-toe postural control (Bastian *et al.*, 1998).

7.2.2. Correlations

Pearson correlations (shown in table 7.5) were calculated in an attempt to examine the interactions of experimental variables across studies. Spearman correlations were also carried out, to guard against the considerable possibility of drawing conclusions based on outliers in small sample Pearson correlations.

7.2.2.1. Balance and EBC

Large reduction in timing error was associated with good stability in the balance alone and balance blindfold tests ($r = 0.636$, $p < 0.05$, $n = 11$; $r = 0.743$, $p < 0.01$, $n = 11$; respectively). Furthermore, large errors in CR timing at block 6 were associated with high wobble values in the balance while counting and balance with fast CRT conditions ($r = 0.644$, $p < 0.05$, $n = 10$; $r = 0.733$, $p < 0.05$, $n = 11$). There was a weaker tendency for large block 6 timing errors to be associated with large wobble scores in the blindfold balance condition ($r = 0.585$, $p = 0.059$, $n = 11$). Significance values were generally reduced in the Spearman analyses but two significant relationships remain, the association between good blindfold balance and good error reduction in CR timing ($r = 0.755$, $p < 0.01$, $n = 11$) and the association between good balance in the balance+fast CRT condition and accurate CR production in block 6 ($r = 0.636$, $p < 0.05$, $n = 11$).

7.2.2.2. Balance and MSA

There were no significant relationships here but two notable trends ($p < 0.1$). High degrees of instability in the balance alone test were associated with impairment in MSA performance at return on day 2 as measured by the simple error-raw speed combination ($r = 0.489$, $p = 0.076$, $n = 14$). The analogous pattern for balance while counting and initial day 2 performance neared statistical significance ($r = 0.543$, $p = 0.055$, $n = 13$). The Spearman tests eliminated the first of these but the second (balance while counting – initial day 2 performance) was reinforced, $r = 0.56$, $p < 0.05$, $n = 13$. Furthermore, a trend for blindfold balance ability to co-occur with initial MSA performance on day 1 emerged in the Spearman analysis ($r = 0.473$, $p = 0.088$, $n = 14$).

Table 7.5. Pearson correlations between key variables in the three studies.

	Balance alone	Blindfold balance	Balance + counting	Balancing + slow CRT	Balancing + fast CRT	CR timing error in block 6	CR timing error reduction from block 2 to block 6	MSA score at pre-1	MSA score at 24hr-1	MSA score at 24hr-4
Balance alone	•									
Blindfold balance	.703 ** 14	•								
Balance + counting	.204 13	.294 13	•							
Balancing + slow CRT	.133 14	.184 14	.826 ** 13	•						
Balancing + fast CRT	.385 14	.698 ** 14	.626 * 13	.453 14	•					
CR timing error in block 6	.162 11	.585 † 11	.644 * 10	.231 11	.733 ** 11	•				
CR timing error reduction from block 2 to block 6	.636 * 11	.743 ** 11	-.138 10	-.318 11	.306 11	.342 11	•			
MSA performance at pre-1	.397 14	.406 14	.133 13	.125 14	.061 14	-.017 11	.686 * 11	•		
MSA performance at 24hr-1	.489 † 14	.331 14	.543 † 13	.356 14	.093 14	.104 11	.317 11	.319 14	•	
MSA performance at 24hr-4	.267 14	.016 14	.301 13	.203 14	-.069 14	-.195 11	.196 11	.292 14	.577 * 14	•

** - $p < 0.01$
* - $p < 0.05$
† - $p < 0.10$

7.2.2.3. EBC and MSA

There was a strong association between good initial performance of the MSA task (trial 1 simple error-raw speed combination) and good improvement of CR timing (from block 2 to block 6), $r = 0.686$, $p < 0.05$, $n = 11$. This relationship was stronger in the Spearman analysis ($r = 0.764$, $p < 0.01$, $n = 11$).

7.2.2.4. Patterns

Consideration of these cross study relationships led to the observation that there were possibly 2 separate groups of related variables. Crudely described the first (balance blindfold, CR timing improvement and initial performance in MSA) involved general motor control and the second (balance while counting, balance during fast CRT, CR timing at block 6 and MSA performance at return on day 2) involved skilled motor performance. To investigate whether these might relate to subgroups within the dyslexic sample the same correlations were examined without the inclusion of control participants. In summary, it could be said that the relationships in the first group of variables (general motor control) remained robust but those in the second (skilled performance) did not. The second group of relationships involving skilled performance may have fallen due to all the skilled performers being in the control group. The relationship (in group 1) between blindfold balance and CR timing error reduction proved particularly resolute ($r = 0.714$, $p < 0.05$, $n = 8$). Consideration of this apparent split produced the idea that the relationships with CR timing improvement might have been a secondary effect of initial timing accuracy, which could be thought of as closer to the global description of 'general motor control' than CR timing improvement. However, further correlational analyses revealed that timing error in block 2 was not significantly related to any of the variables in this 'general motor control' group of variables.

8. Concluding discussion

This study set out to examine adult participants with enduring evidence of dyslexia, in order to discover whether deficits in areas outside of literacy also endured into adulthood, and by implication, whether there were grounds to assume that these impairments were also fundamental to the character of dyslexia. A range of data were collected including measures of IQ, reading, writing, spelling, attention deficit disorder, balance, postural stability, gross motor adaptation, classical conditioning of eyeblinks and the learning and consolidation of a sequence of finger movements.

The following conclusions can be drawn from the research reported in this thesis:

8.1.1.1. Balance

There are problems in the gross motor skills of balance and postural control in dyslexia. Difficulties in balance and postural reflex adaptation are most pronounced when conscious compensation is prevented using a dual-task paradigm. Particular difficulty in the heel-to-toe posture may be a result of sub-optimal cross-hemispheric transfer in the cerebellar cortex, since that posture was most severely impaired in 5 children who underwent transection of the posterior inferior cerebellar vermis (Bastian *et al.*, 1998). Inappropriate methodology could easily mask balance problems in dyslexia.

8.1.1.2. EBC

Of those who passed the criterion of 11 or more conditioned responses in 54 trials, participants with dyslexia were much less likely to tune their conditioned responses appropriately, to coincide with airpuff (unconditioned stimulus) delivery. Hence, in parallel with the balance findings, dyslexics failed to produce the optimized motor performance seen in the control group. This in a task known to critically rely on the cerebellum. Hence this study provides a second line of converging evidence indicating a general failure to optimize motor performance that could be caused by cerebellar abnormality.

8.1.1.3. MSA

The study of motor skill acquisition showed that dyslexics learned to perform a simple sequence of finger movements as well as control participants did, both when the task was novel and when explicitly rehearsing the sequence. In contrast, as a whole the dyslexic group did not perform as well as controls after a night's sleep. Sleep is crucial for the consolidation of such learning (Korman *et al.*, 2003; Walker *et al.*, 2002) and produces an altered pattern of brain activity during task performance (with notable increases in activation in primary motor cortex, medial pre-frontal lobe, hippocampus and cerebellum (Walker *et al.*, 2005). Incomplete consolidation of memories could be expected to lead to difficulty in automatization

of skill, which is well documented in dyslexia (Moores *et al.*, 2003; Nicolson & Fawcett, 1990) and indeed was demonstrated in chapter 4 of this thesis. However, the extent of the role of the cerebellum in this type of learning is not clear. At least one prominent theory (Doyon & Benali, 2005) has suggested that the cerebellum is only important in the earlier stages of motor skill acquisition and is less important (at least in the long-term) than the striatum and cerebral cortex. However, abnormal learning processes in the early stages whilst performance is ostensibly normal (perhaps due to extra effort on the part of dyslexic subjects) might still contribute to consolidation impairment.

8.1.1.4. Implications for theoretical approaches

Evidence of deficits outside the literacy domain is problematic for adherents of a pure phonological deficit view of dyslexia since the PDH does not predict these deficits. Furthermore, as discussed in the individual chapters, it is difficult to explain the findings of this thesis in terms of processing speed/sensory input problems. So while the predictions of the double deficit and magnocellular deficit approaches are broader than those of the PDH, they do not fit so comfortably with data presented here are the predictions of the cerebellar theory do. The cerebellar/automatization deficit framework predicts the deficits in balance, classical conditioning and motor learning reported here.

8.1.1.5. ADD

There is a higher incidence of ADD in dyslexia than in the general population. The cause of this is not known, but two outstanding possibilities are that (i) *The ADD scale used partly tapped dyslexia itself.* Particularly the 2 sections (of 5 in total) on memory and emotion would be expected to produce high scores for people who were 'purely' dyslexic, since poor memory (McLoughlin et al., 1994) and low self-esteem (McNulty, 2003) are frequently reported in such individuals. And, (ii) *Dyslexia and ADD share common or overlapping biological aetiology.* For example, both are associated with cerebellar abnormality (Eckert *et al.*, 2003; Seidman *et al.*, 2005). In general, the exclusion of persons with ADD did not alter the pattern of results. The most notable exception was that the dyslexic participants seemed to progress better on the explicit stages of the MSA experiment when those with high ADD scores were excluded from analyses. Importantly, signs of ADD seem unable to explain balance deficits in dyslexic in the present sample.

8.1.1.6. Heterogeneity

Dyslexia is highly heterogeneous - large proportions of the present dyslexic samples showed considerable impairment in the three respective studies, but none of these sets of experiments caused particular difficulty for all dyslexic participants. This could be explained by the idea, that the syndrome we refer to as "dyslexia" is caused by overlapping but not identical biological abnormalities, with the common behavioural product of reading failure being the most frequently noticed in a society

that relies so heavily on (and indeed often assumes) reading competence by adulthood.

8.1.1.7. MSA and the cerebellum

Of those providing data for all studies, all of the dyslexic participants who showed impairment in balance, EBC or MSA showed impairment in at least balance or EBC. That is to say that none had MSA deficit alone. Only one of the eight had impairment in none. Consequently a more minor role for the cerebellum in MSA than in balance and EBC certainly fits with the cross-study impairment prevalence data presented in section 7.2.1. of this thesis as well as with the background literature.

8.1.1.8. Two types of motor ability?

Two strands of motor impairment have been alluded to throughout this thesis, (i) *poor general motor skill*, the phenomenon of producing relatively poor motor performance in all motor skill areas, presumably due to having globally less precise control of one's limbs, or possibly low processing speed. And (ii) *poor motor skill acquisition*, more specifically the inability to unconsciously improve one's performance, which could mean failure to automatize balance, adaptively time classically conditioned eyeblinks or consolidate the procedural memory of a learned motor sequence while asleep. Analysis of the data generated by participants who completed all the experiments suggests that these two phenomena may be separable and distinct. This claim should be treated with caution as it is based on a particularly small sample, but it certainly merits further investigation. If the distinction proves robust then it is possible that different individuals could be impaired in one or other or both of these areas, in a way analogous to Wolf and Bowers' analysis of processing speed and phonology in their Double Deficit hypothesis (1999). Were this the case then clearly the most effective interventions would be targeted according to the particular locus of the problem in each specific case.

8.1.1.9. Replication needed

Motor skill acquisition deficits in dyslexia demand more research attention. If these results were replicated in larger samples there would be strong implications for the future of the field. It would be particularly interesting to see whether the patterns reported here are replicable (with appropriate methodological adaptations) in a pre-school sample. Clearly such participants could not be diagnosed dyslexics at the time of testing, however the sample could be followed up later in life with tests of literacy attainment. Family histories or genetic analyses together with a pre-school screening test for dyslexia (Fawcett *et al.*, 2001a) could be used to increase the chance of the sample including a high proportion of participants likely to struggle with reading.

8.1.1.10. An area for further research

The notion of different but overlapping areas of underlying biological (possibly cerebellar) abnormality also deserves more attention. An imaging study assessing areas of cerebellar activity in motor and reading tasks, in dyslexic-motor impaired, dyslexic motor-unimpaired and non-dyslexic participants would no doubt be very informative. If the dyslexic groups shared a common pattern of activation during reading tests that differed from the control group pattern, but then the dyslexic motor-unimpaired group showed activation similar to controls in motor tasks but different to the dyslexic motor-impaired group, it would be of interest to compare the areas of difference between the groups across the two sets of experiments. If differences in activation were found between the dyslexic motor-impaired group and the control group in reading tasks that were proximal to sites of discrepancy between the dyslexic groups in motor tasks then the idea that heterogeneity of motor skill in dyslexia was attributable to small differences in biological location of dysfunction would gain weight.

8.1.1.11. Limitations

The work presented in this thesis is not without its limitations.

a. Firstly I should acknowledge again that the small sample size for the cross study comparisons (8 dyslexic and 6 control) has weakened that section. Whilst it has been informative to undertake and has certainly made the case for the future adoption of this approach in future research, the specific conclusions should be treated with some caution until they are replicated.
b. The large proportion of EBC participants failing to make the CR criterion is a cause for concern, again this limited the sample size for analyses of CR timing. However, the finding of normal strength but impaired accuracy of responding in that study is firmly in agreement with the earlier experiments (Nicolson *et al.*, 2002).
c. The sensory organisation test (SOT) seemed insufficiently sensitive for the detection of between group differences, with both groups performing close to ceiling in all dual-task conditions.
d. Representativeness of the sample. The participants in this study were mostly students at the University of Sheffield, and as such were generally young and well educated. Furthermore they were diagnosed according to this university's particular criteria, as we have seen, dyslexia definition varies widely from one institution to the next.

8.2. Final conclusion

The studies described here provide evidence of difficulties for those with dyslexia in three different areas of performance and learning outside the domain of literacy. The general pattern of results is most consistent with the cerebellar deficit hypothesis. Although there was evidence of co-morbid ADD in a large proportion of dyslexic participants the learning and performance deficits observed appeared not to be the

result of this co-morbidity. Individual analyses showed that group deficits in balance, postural control, eyeblink conditioning and motor skill acquisition were attributable to a subset of participants in each study. There was some consistency of within participant performance across the studies, with balance and eyeblink conditioning perhaps more closely linked to each other than to the motor skill acquisition task. Owing particularly to the small sample size for the cross study analyses, further research on this model is recommended towards clarity on the consistency of motor learning and performance deficits in dyslexia.

9: References

Ackermann, H., & Hertrich, I. (2000). The contribution of the cerebellum to speech processing. *Journal of Neurolinguistics, 13*, 95-116.

Albus, J. S. (1971). A theory of cerebellar function. *Mathematical Biosciences, 10*, 25-61.

Badian, N. A. (1984). Reading disability in an epidemiological context: Incidence and environmental correlates. *Journal of Learning Disabilities, 17*(3), 129-136.

Bao, S. W., Chen, L., Kim, J. J., & Thompson, R. F. (2002). Cerebellar cortical inhibition and classical eyeblink conditioning. *Proceedings of the National Academy of Sciences of the United States of America, 99*(3), 1592-1597.

Bastian, A. J., Mink, J. W., Kaufman, B. A., & Thach, W. T. (1998). Posterior vermal split syndrome. *Annals Of Neurology, 44*(4), 601-610.

Beaton, A. A. (2002). Dyslexia and the cerebellar deficit hypothesis. *Cortex, 38*, 479-490.

Biscaldi, M., Fischer, B., & Hartnegg, K. (2000). Voluntary saccadic control in dyslexia. *Perception, 29*(5), 509-521.

Biscaldi, M., Gezeck, S., & Stuhr, V. (1998). Poor saccadic control correlates with dyslexia. *Neuropsychologia, 36*(11), 1189-1202.

Bower, J. M., & Parsons, L. M. (2003). Rethinking the "lesser brain". *Scientific American, 289*, 50-57.

Bradley, L., & Bryant, P. E. (1983). Categorising sounds and learning to read: A causal connection. *Nature, 301*, 419-421.

Brookes, R. L., & Stirling, J. (2005). The cerebellar deficit hypothesis and dyslexic tendencies in a non-clinical sample. *Dyslexia, 11*(3), 174-185.

Brown, T. E. (1996). Brown attention-deficit disorder scales. *Psychological Corporation, Harcourt Brace & Company.*

Catts, H. W. (1996). Defining dyslexia as a developmental language disorder - an expanded view. *Topics In Language Disorders, 16*(2), 14-29.

Christian, K. M., & Thompson, R. F. (2003). Neural substrates of eyeblink conditioning: Acquisition and retention. *Learning & Memory, 10*(6), 427-455.

Coffin, J. M., Baroody, S., Schneider, K., & O'Neill, J. (2005). Impaired cerebellar learning in children with prenatal alcohol exposure: A comparative study of eyeblink conditioning in children with adhd and dyslexia. *Cortex, 41*(3), 389-398.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences (2nd ed).* New York: Academic Press.

Cornelissen, P., Richardson, A., Mason, A., Fowler, S., & Stein, J. F. (1995). Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls. *Vision Research, 35*, 1483-1494.

Daum, I., Schugens, M. M., Ackermann, H., Lutzenberger, W., Dichgans, J., & Birbaumer, N. (1993). Classical conditioning after cerebellar lesions in humans. *Behavioural Neuroscience, 107*(5), 748-756.

Demonet, J. F., Taylor, M. J., & Chaix, Y. (2004). Developmental dyslexia. *Lancet, 363*(9419), 1451-1460.

Denckla, M. B., & Rudel, R. G. (1976). Rapid 'automatized' naming (R.A.N.). *Dyslexia differentiated from other learning disabilities. Neuropsychologia, 14*, 471-479.

Desmond, J. E., & Fiez, J. A. (1998). Neuroimaging studies of the cerebellum: Language, learning and memory. *Trends in Cognitive Sciences, 2*(9), 355-362.

Dimitrova, A., Weber, J., Maschke, M., Elles, H. G., Kolb, F. P., Forsting, M., et al. (2002). Eyeblink-related areas in human cerebellum as shown by fMRI. *Human Brain Mapping, 17*(2), 100-115.

Dow, R. S., & Moruzzi, G. (1958). *The physiology and pathology of the cerebellum.* Minneapolis: University of Minnesota Press.

Doya, K. (2000). Complementary roles of basal ganglia and cerebellum in learning and motor control. *Current Opinion in Neurobiology, 10*(6), 732-739.

Doyon, J., & Benali, H. (2005). Reorganization and plasticity in the adult brain during learning of motor skills. *Current Opinion In Neurobiology, 15*(2), 161-167.

Doyon, J., Penhune, V., & Ungerleider, L. G. (2003). Distinct contribution of the cortico-striatal and cortico- cerebellar systems to motor skill learning. *Neuropsychologia, 41*(3), 252-262.

Eckert, M. A., Leonard, C. M., Richards, T. L., Aylward, E. H., Thomson, J., & Berninger, V. W. (2003). Anatomical correlates of dyslexia: Frontal and cerebellar findings. *Brain, 126*, 482-494.

Eckert, M. A., Leonard, C. M., Wilke, M., Eckert, M., Richards, T., Richards, A., et al. (2005). Anatomical signatures of dyslexia in children: Unique information from manual and voxel based morphometry brain measures. *Cortex, 41*(3), 304-315.

Eden, G. F., Jones, K. M., Cappell, K., Gareau, L., Wood, F. B., Zeffiro, T. A., et al. (2004). Neural changes following remediation in adult developmental dyslexia. *Neuron, 44*(3), 411-422.

Eden, G. F., VanMeter, J. W., Rumsey, J. M., Maisog, J. M., Woods, R. P., & Zeffiro, T. A. (1996). Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature, 382*, 66-69.

Fabbro, F. (2000). Introduction to language and cerebellum. *Journal of Neurolinguistics, 13*(2-3), 83-94.

Facoetti, A., Turatto, M., Lorusso, M. L., & Mascetti, G. G. (2001). Orienting of visual attention in dyslexia: Evidence for asymmetric hemispheric control of attention. *Experimental Brain Research, 138*(1), 46-53.

Fawcett, A. J., & Nicolson, R. I. (1992). Automatisation deficits in balance for dyslexic children. *Perceptual and Motor Skills, 75*(2), 507-529.

Fawcett, A. J., & Nicolson, R. I. (1994). Naming speed in children with dyslexia. *Journal of Learning Disabilities, 27*(10), 641-646.

Fawcett, A. J., & Nicolson, R. I. (1995). Persistent deficits in motor skill of children with dyslexia. *Journal of Motor Behavior, 27*(3), 235-240.

Fawcett, A. J., & Nicolson, R. I. (1996). *The dyslexia screening test.* London: The Psychological Corporation.

Fawcett, A. J., & Nicolson, R. I. (1998). *Dyslexia adult screening test.* London, UK: The Psychological Corporation.

Fawcett, A. J., & Nicolson, R. I. (1999). Performance of dyslexic children on cerebellar and cognitive tests. *Journal Of Motor Behavior, 31*(1), 68-78.

Fawcett, A. J., & Nicolson, R. I. (2001). Dyslexia: The role of the cerebellum. In A. J. Fawcett (Ed.), *Dyslexia theory and good practice.* London: Whurr.

Fawcett, A. J., & Nicolson, R. I. (2002). Children with dyslexia are slow to articulate a single speech gesture. *Dyslexia, 8,* 189-203.

Fawcett, A. J., Nicolson, R. I., & Lee, R. (2001a). *Pre-school screening test.* London: The Psychological Corporation.

Fawcett, A. J., Nicolson, R. I., & Maclagan, F. (2001b). Cerebellar tests differentiate between groups of poor readers with and without iq discrepancy. *Journal of Learning Disabilities, 34*(2), 119-135.

Finucci, J. M., Guthrie, J. T., Childs, A. L., Abbey, H., & Childs, B. (1976). The genetics of specific reading disability. *Annals of Human Genetics, 50*(1-23).

Fischer, B., Hartnegg, K., & Mokler, A. (2000). Dynamic visual perception of dyslexic children. *Perception, 29*(5), 523-530.

Fisher, S. E., & Smith, S. D. (2001). Progress towards the identification of genes influencing developmental dyslexia. In A. Fawcett (Ed.), *Dyslexia theory and good practice* (pp. 39-64). London: Whurr.

Frith, U. (1997). Brain, mind and behaviour in dyslexia. In C. Hulme & M. Snowling (Eds.), *Dyslexia: Biology, cognition and intervention.* London: Whurr.

Fulbright, R. K., Jenner, A. R., Mencl, W. E., Pugh, K. R., Shaywitz, B. A., Shaywitz, S. E., et al. (1999). The cerebellum's role in reading: A functional mr imaging study. *American Journal of Neuroradiology, 20*(10), 1925-1930.

Galaburda, A. M. (1999). Developmental dyslexia: A multilevel syndrome. *Dyslexia, 5,* 183-191.

Galaburda, A. M., Rosen, G. D., & Sherman, G. F. (1989). The neural origin of developmental dyslexia: Implications for medicine, neurology and cognition. In A. M. Galaburda (Ed.), *From reading to neurons.* Cambridge, MA: MIT Press.

Garcia, K. S., & Mauk, M. D. (1998). Pharmacological analysis of cerebellar contributions to the timing and expression of conditioned eyelid responses. *Neuropharmacology, 37*(4-5), 471-480.

Gerwig, M., Dimitrova, A., Kolb, F. P., Maschke, M., Brol, B., Kunnel, A., et al. (2003). Comparison of eyeblink conditioning in patients with superior and posterior inferior cerebellar lesions. *Brain, 126,* 71-94.

Gerwig, M., Dimitrova, A., Maschke, M., Kolb, F. P., Forsting, M., & Timmann, D. (2004). Amplitude changes of unconditioned eyeblink responses in patients with cerebellar lesions. *Experimental Brain Research, 155*(3), 341-351.

Gluck, M. A., Allen, M. T., Myers, C. E., & Thompson, R. F. (2001). Cerebellar substrates for error correction in motor conditioning. *Neurobiology of Learning and Memory, 76*(3), 314-341.

Green, J. T., Ivry, R. B., & Woodruff-Pak, D. S. (1999). Timing in eyeblink classical conditioning and timed-interval tapping. *Psychological Science, 10*(1), 19-23.

Hesslow, G., & Yeo, C. H. (2002). The functional anatomy of skeletal conditioning. In J. W. Moore (Ed.), *A neuroscientist's guide to classical conditioning* (pp. 86-146). New York: Springer-Verlag.

Hetrick, W. P., Brown, S. M., Kieffaber, P. D., Vohs, J. L., Carroll, C. A., O'Donnell, B. F., et al. (2004). Eyeblink conditioning deficits indicate timing abnormalities in schizophrenia. *Integrative Physiological and Behavioral Science, 39*(1), 58-58.

Holmes, G. (1917). The symptoms of acute cerebellar injuries due to gunshot injuries. *Brain, 40*, 461-535.

Hulslander, J., Talcott, J., Witton, C., DeFries, J., Pennington, B., Wadsworth, S., et al. (2004). Sensory processing, reading, iq, and attention. *Journal of Experimental Child Psychology, 88*(3), 274-295.

Imamizu, H., Miyauchi, S., Tamada, T., Sasaki, Y., Takino, R., Putz, B., et al. (2000). Human cerebellar activity reflecting an acquired internal model of a new tool. *Nature, 403*(6766), 192-195.

Ivry, R. (2000). Exploring the role of the cerebellum in sensory anticipation and timing: Commentary on tesche and karhu. *Human Brain Mapping, 9*(3), 115-118.

Ivry, R., & Keele, S. W. (1989). Timing functions of the cerebellum. *Journal of Cognitive Neuroscience, 1*, 136-152.

Jenkins, I. H., Brooks, D. J., Nixon, P. D., Frackowiak, R. S. J., & Passingham, R. E. (1994). Motor sequence learning - a study with positron emission tomography. *Journal Of Neuroscience, 14*(6), 3775-3790.

Jorm, A. F., Share, D. L., McLean, R., & Matthews, D. (1986). Cognitive factors at school entry predictive of specific reading retardation and general reading backwardness: A research note. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 27*, 45-54.

Kaplan, B. J., Dewey, D. M., Crawford, S. G., & Wilson, B. N. (2001). The term comorbidity is of questionable value in reference to developmental disorders: Data and theory. *Journal of Learning Disabilities, 34*(6), 555-565.

Karni, A., Meyer, G., Jezzard, P., Adams, M., Turner, R., & Ungerleider, L. G. (1995). Functional mri evidence for adult motor cortex plasticity during motor skill learning. *Nature, 377*(6545), 155-158.

Kleim, J. A., Hogg, T. M., VandenBerg, P. M., Cooper, N. R., Bruneau, R., & Remple, M. (2004). Cortical synaptogenesis and motor map reorganization occur during late, but not early, phase of motor skill learning. *Journal Of Neuroscience, 24*(3), 628-633.

Korman, M., Raz, N., Flash, T., & Karni, A. (2003). Multiple shifts in the representation of a motor sequence during the acquisition of skilled

performance. *Proceedings of the National Academy of Sciences of the United States of America, 100*(21), 12492-12497.

Lang, C. E., & Bastian, A. J. (2002). Cerebellar damage impairs automaticity of a recently practiced movement. *Journal of Neurophysiology, 87*(3), 1336-1347.

Leiner, H. C., Leiner, A. L., & Dow, R. S. (1993). Cognitive and language functions of the human cerebellum. *Trends in Neurosciences, 16*(11), 444-447.

Livingstone, M. S., Rosen, G. D., Drislane, F. W., & Galaburda, A. M. (1993). Physiological and anatomical evidence for a magnocellular deficit in developmental dyslexia. Correction. *Proceedings of the National Academy of Sciences of the USA, 90*, 2256.

Lovegrove, W. J., Bowling, A., Badcock, B., & Blackwood, M. (1980). Specific reading disability: Differences in contrast sensitivity as a function of spatial frequency. *Science, 210*, 439-440.

Lyon, G. R., Shaywitz, S. E., & Shaywitz, B. A. (2003). A definition of dyslexia. *Annals Of Dyslexia, 53*, 1-14.

MacDonnell, L. E. F., Skinner, F. K., Eward, P. E., Glen, A. I. M., Glen, A. C. A., Macdonald, D. J., et al. (2000). Increased levels of cytosolic phospholipase a2 in dyslexics. *Prostaglandins Leukotrienes And Essential Fatty Acids, 63*(1-2), 37-39.

Makris, N., Hodge, S. M., Haselgrove, C., Kennedy, D. N., Dale, A., Fischl, B., et al. (2003). Human cerebellum: Surface-assisted cortical parcellation and volumetry with magnetic resonance imaging. *Journal of Cognitive Neuroscience, 15*(4), 584-599.

Marr, D. (1969). A theory of cerebellar cortex. *Journal Of Physiology-London, 202*(2), 437-&.

Martin, J., & Lovegrove, W. (1987). Flicker contrast sensitivity in normal and specifically-disabled readers. *Perception, 16*, 215-221.

McLoughlin, D., Fitzgibbon, G., & Young, V. (1994). *Adult dyslexia: Assessment, counselling and training.* London: Whurr.

McNulty, M. A. (2003). Dyslexia and the life course. *Journal Of Learning Disabilities, 36*(4), 363-381.

Moe-Nilssen, R., Helbostad, J. L., Talcott, J. B., & Toennessen, F. E. (2003). Balance and gait in children with dyslexia. *Experimental Brain Research, 150*(2), 237-244.

Moores, E., Nicolson, R., & Fawcett, A. (2003). Attention deficits in dyslexia: Evidence for an automatisation deficit? *European Journal Of Cognitive Psychology, 15*(3), 321-348.

Moretti, R., Bava, A., Torre, P., Antonello, R. M., & Cazzato, G. (2002). Reading errors in patients with cerebellar vermis lesions. *Journal of Neurology, 249*(4), 461-468.

Needle, J. L., Fawcett, A. J., & Nicolson, R. I. (in press). Balance and dyslexia: An investigation of adults' abilities. *European Journal of Cognitive Psychology.*

Nicolson, R. I. (2001). Developmental dyslexia - into the future. In A. J. Fawcett (Ed.), *Dyslexia theory and good practice* (pp. 1-35). London: Whurr.

Nicolson, R. I., Daum, I., Schugens, M. M., Fawcett, A. J., & Schulz, A. (2002). Eyeblink conditioning indicates cerebellar abnormality in dyslexia. *Experimental Brain Research, 143*(1), 42-50.

Nicolson, R. I., & Fawcett, A. J. (1990). Automaticity: A new framework for dyslexia research. *Cognition, 35*, 159-182.

Nicolson, R. I., & Fawcett, A. J. (1994a). Comparison of deficits in cognitive and motor-skills among children with dyslexia. *Annals of Dyslexia, 44*, 147-164.

Nicolson, R. I., & Fawcett, A. J. (1994b). Reaction-times and dyslexia. *Quarterly Journal of Experimental Psychology Section a-Human Experimental Psychology, 47*(1), 29-48.

Nicolson, R. I., & Fawcett, A. J. (1995). Dyslexia is more than a phonological disability. *Dyslexia: An international journal of research and practice, 1*, 19-37.

Nicolson, R. I., & Fawcett, A. J. (1996). *The dyslexia early screening test*. London: The Psychological Corporation.

Nicolson, R. I., & Fawcett, A. J. (1997). Development of objective procedures for screening and assessment of dyslexic students in higher education. *Journal of Research in Reading, 20*(1), 77-83.

Nicolson, R. I., & Fawcett, A. J. (2000). Long-term learning in dyslexic children. *European Journal of Cognitive Psychology, 12*(3), 357-393.

Nicolson, R. I., & Fawcett, A. J. (Submitted). *Developmental dyslexia: Learning, cognition and the brain*. Massachusetts: MIT press.

Nicolson, R. I., Fawcett, A. J., Berry, E. L., Jenkins, I. H., Dean, P., & Brooks, D. J. (1999). Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. *Lancet, 353*(9165), 1662-1667.

Nicolson, R. I., Fawcett, A. J., & Dean, P. (1995). Time-estimation deficits in developmental dyslexia - evidence of cerebellar involvement. *Proceedings of the Royal Society of London Series B-Biological Sciences, 259*(1354), 43-47.

Nicolson, R. I., Fawcett, A. J., & Dean, P. (2001). Developmental dyslexia: The cerebellar deficit hypothesis. *Trends in Neurosciences, 24*(9), 508-511.

Ohno, H., Unushihara, R., Sei, H., & Morita, Y. (2002). Rem sleep deprivation suppresses acquisition of classical eyeblink conditioning. *Sleep, 25*(8), 877-881.

Ohyama, T., & Mauk, M. D. (2001). Latent acquisition of timed responses in cerebellar cortex. *Journal of Neuroscience, 21*(2), 682-690.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia, 9*, 97-113.

Ouchi, Y., Okada, H., Yoshikawa, E., Nobezawa, S., & Futatsubashi, M. (1999). Brain activation during maintenance of standing postures in humans. *Brain, 122*, 329-338.

Pavlov, I. P. (1927). *Conditioned reflexes*. Oxford: Oxford University Press.

Poblano, A., Ishiwara, K., Arias, M. D., Garcia-Pedroza, F., Marin, H., & Trujillo, M. (2002). Motor control alteration in posturography in learning-disabled children. *Archives of Medical Research, 33*(5), 485-488.

Raberger, T., & Wimmer, H. (2003). On the automaticity/cerebellar deficit hypothesis of dyslexia: Balancing and continuous rapid naming in dyslexic and adhd children. *Neuropsychologia, 41*(11), 1493-1497.

Rack, J. (2003). The who, what, why and how of intervention programmes: Comments on the ddat evaluation. *Dyslexia, 9*(3), 137-139.

Rae, C., Harasty, J. A., Dzendrowskyj, T. E., Talcott, J. B., Simpson, J. M., Blamire, A. M., et al. (2002). Cerebellar morphology in developmental dyslexia. *Neuropsychologia, 40*(8), 1285-1292.

Rae, C., Lee, M. A., Dixon, R. M., Blamire, A. M., Thompson, C. H., Styles, P., et al. (1998). Metabolic abnormalities in developmental dyslexia detected by h-1 magnetic resonance spectroscopy. *Lancet, 351*(9119), 1849-1852.

Ramnani, N., Toni, I., Josephs, O., Ashburner, J., & Passingham, R. E. (2000). Learning- and expectation-related changes in the human brain during motor learning. *Journal of Neurophysiology, 84*(6), 3026-3035.

Ramus, F. (2003). Developmental dyslexia: Specific phonological deficit or general sensorimotor dysfunction? *Current Opinion in Neurobiology, 13*(2), 212-218.

Ramus, F. (2004). Neurobiology of dyslexia: A reinterpretation of the data. *Trends In Neurosciences, 27*(12), 720-726.

Ramus, F., Pidgeon, E., & Frith, U. (2003a). The relationship between motor control and phonology in dyslexic children. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 44*(5), 712-722.

Ramus, F., Rosen, S., Dakin, S. C., Day, B. L., Castellote, J. M., White, S., et al. (2003b). Theories of developmental dyslexia: Insights from a multiple case study of dyslexic adults. *Brain, 126*, 841-865.

Rapoport, M., van Reekum, R., & Mayberg, H. (2000). The role of the cerebellum in cognition and behavior: A selective review. *Journal of Neuropsychiatry and Clinical Neurosciences, 12*(2), 193-198.

Reynolds, D., Nicolson, R. I., & Hambly, H. (2003). Evaluation of an exercise-based treatment for children with reading difficulties. *Dyslexia, 9*(1), 48-71.

Riva, D. (2000). Cerebellar contribution to behaviour and cognition in children. *Journal of Neurolinguistics, 13*(2-3), 215-225.

Rogers, R. F., Britton, G. B., & Steinmetz, J. E. (2001). Learning-related interpositus activity is conserved across species as studied during eyeblink conditioning in the rat. *Brain Research, 905*(1-2), 171-177.

Rogers, R. F., Fender, A. F., & Steinmetz, J. E. (1999). The cerebellum is necessary for rabbit classical eyeblink conditioning with a non-somatosensory (photic) unconditioned stimulus. *Behavioural Brain Research, 104*(1-2), 105-112.

Rumsey, J. M., Horwitz, B., Donohue, B. C., Nace, K. L., Maisog, J. M., & Andreason, P. (1999). A functional lesion in developmental dyslexia: Left angular gyral blood flow predicts severity. *Brain and Language, 70*, 187-204.

Sakai, K., Ramnani, N., & Passingham, R. E. (2002). Learning of sequences of finger movements and timing: Frontal lobe and action-oriented representation. *Journal of Neurophysiology, 88*(4), 2035-2046.

Schreurs, B. G., Bahro, M., Molchan, S. E., Sunderland, T., & McIntosh, A. R. (2001). Interactions of prefrontal cortex during eyeblink conditioning as a function of age. *Neurobiology of Aging, 22*(2), 237-246.

Sears, L. L., Finn, P. R., & Steinmetz, J. E. (1994). Abnormal classical eye-blink conditioning in autism. *Journal of Autism and Developmental Disorders, 24*(6), 737-751.

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

Seki, A., Okada, T., Koeda, T., & Sadato, N. (2004). Phonemic manipulation in Japanese: An fMRI study. *Cognitive Brain Research, 20*(2), 261-272.

Shadmehr, R., & Holcomb, H. H. (1997). Neural correlates of motor memory consolidation. *Science, 277*(5327), 821-825.

Shaywitz, B. A., Shaywitz, S. E., Pugh, K. R., Mencl, W. E., Fulbright, R. K., Skudlarski, P., et al. (2002). Disruption of posterior brain systems for reading in children with developmental dyslexia. *Biological Psychiatry, 52*(2), 101-110.

Shaywitz, S. (1996). Dyslexia. *Scientific American*(November), 78-84.

Shaywitz, S. E. (1998). Current concepts - dyslexia. *New England Journal of Medicine, 338*, 307-312.

Shaywitz, S. E., Fletcher, J. M., & Shaywitz, B. A. (1994). Issues in the definition and classification of attention- deficit disorder. *Topics In Language Disorders, 14*(4), 1-25.

Shea, C. H., Lai, Q., Black, C., & Park, J. H. (2000). Spacing practice sessions across days benefits the learning of motor skills. *Human Movement Science, 19*(5), 737-760.

Siegel, L. S. (1989). Why we do not need intelligence test scores in the definition and analyses of learning disabilities. *Journal of Learning Disabilities, 22*, 514-518.

Silveri, M. C., & Misciagna, S. (2000). Language, memory, and the cerebellum. *Journal of Neurolinguistics, 13*(2-3), 129-143.

Snowling, M. J. (1981). Phonemic deficits in developmental dyslexia. *Psychological Research, 43*, 219–234.

Snowling, M. J. (1995). Phonological processing and developmental dyslexia. *Journal of Research in Reading, 18*, 132-138.

Snowling, M. J., & Hulme, C. (2003). A critique of claims from Reynolds, Nicolson & Hambly (2003) that ddat is an effective treatment for children with reading difficulties - 'lies, damned lies and (inappropriate) statistics?' *Dyslexia, 9*(2), 127-133.

Stanovich, K. E. (1988). Explaining the differences between the dyslexic and the garden-variety poor reader: The phonological-core variable-difference model. *Journal of Learning Disabilities, 21*(10), 590-604.

Stanovich, K. E., & Siegel, L. S. (1994). The phenotypic performance profile of reading-disabled children: A regression-based test of the phonological-core variable-difference model. *Journal of Educational Psychology, 86*, 24-53.

Stein, J. F. (2001). The magnocellular theory of developmental dyslexia. *Dyslexia, 7*, 12-36.

Stein, J. F., Talcott, J., & Witton, C. (2001). The sensorimotor basis of developmental dyslexia. In A. Fawcett (Ed.), *Dyslexia theory and good practice* (pp. 65-88). London: Whurr.

Stein, J. F., & Walsh, V. (1997). To see but not to read; the magnocellular theory of dyslexia. *Trends In Neurosciences, 20*(4), 147-152.

Steinmetz, J. E. (2000). Brain substrates of classical eyeblink conditioning: A highly localized but also distributed system. *Behavioural Brain Research, 110*(1-2), 13-24.

Steinmetz, J. E., Tracy, J. A., & Green, J. T. (2001). Classical eyeblink conditioning: Clinical models and applications. *Integrative Physiological and Behavioral Science, 36*(3), 220-238.

Stoodley, C. J., Fawcett, A. J., Nicolson, R. I., & Stein, J. F. (2005). Impaired balancing ability in dyslexic children. *Experimental Brain Research, published online.*

Stoodley, C. J., Talcott, J. B., Carter, E. L., Witton, C., & Stein, J. F. (2000). Selective deficits of vibrotactile sensitivity in dyslexic readers. *Neuroscience Letters, 295*(1-2), 13-16.

Takatsuki, K., Kawahara, S., Kotani, S., Fukunaga, S., Mori, H., Mishina, M., et al. (2003). The hippocampus plays an important role in eyeblink conditioning with a short trace interval in glutamate receptor subunit delta 2 mutant mice. *Journal of Neuroscience, 23*(1), 17-22.

Talcott, J. B., Gram, A., Van Ingelghem, M., Witton, C., Stein, J. F., & Toennessen, F. E. (2003). Impaired sensitivity to dynamic stimuli in poor readers of a regular orthography. *Brain and Language, 87*(2), 259-266.

Talcott, J. B., Hansen, P. C., Assoku, E. L., & Stein, J. F. (2000). Visual motion sensitivity in dyslexia: Evidence for temporal and energy integration deficits. *Neuropsychologia, 38*(7), 935-943.

Talcott, J. B., Hansen, P. C., Willis-Owen, C., McKinnell, I. W., Richardson, A. J., & Stein, J. F. (1998). Visual magnocellular impairment in adult developmental dyslexics. *Neuro-Ophthalmology, 20*(4), 187-201.

Tallal, P., Miller, S., & Fitch, R. H. (1993). Neurobiological basis of speech - a case for the pre-eminence of temporal processing. *Annals of the New York Academy of Sciences, 682*, 27-47.

Tallal, P., & Piercy, M. (1973). Defects of non-verbal auditory perception in children with developmental aphasia. *Nature, 241*(468-9).

Temple, E., Poldrack, R. A., Protopapas, A., Nagarajan, S., Salz, T., Tallal, P., et al. (2000). Disruption of the neural response to rapid acoustic stimuli in dyslexia: Evidence from functional mri. *Proceedings of the National Academy of Sciences of the United States of America, 97*(25), 13907-13912.

Thach, W. T. (1998). What is the role of the cerebellum in motor learning and cognition? *Trends In Cognitive Sciences, 2*(9), 331-337.

Toni, I., Krams, M., Turner, R., & Passingham, R. E. (1998). The time course of changes during motor sequence learning: A whole-brain fMRI study. *Neuroimage, 8*(1), 50-61.

Torgesen, J. K., Wagner, R. K., Rashotte, C. A., Rose, E., Lindamood, P., Conway, T., et al. (1999). Preventing reading failure in young children with phonological processing disabilities: Group and individual responses to instruction. *Journal of Educational Psychology, 91*(4), 579-593.

Tracy, J. A., Ghose, S. S., Stecher, T., McFall, R. M., & Steinmetz, J. E. (1999). Classical conditioning in a nonclinical obsessive-compulsive population. *Psychological Science, 10*(1), 9-13.

Tracy, J. I., Faro, S. S., Mohammed, F., Pinus, A., Christensen, H., & Burkland, D. (2001). A comparison of 'early' and 'late' stage brain activation during brief practice of a simple motor task. *Cognitive Brain Research, 10*(3), 303-316.

Ullman, M. T. (2004). Contributions of memory circuits to language: The declarative/procedural model. *Cognition, 92*(1-2), 231-270.

van Daal, V., & van der Leij, A. (1999). Developmental dyslexia: Related to specific or general deficits? *Annals Of Dyslexia, 49*, 71-104.

Vellutino, F. R., Fletcher, J. M., Snowling, M., & Scanlon, D. M. (2004). Specific reading disability (dyslexia): What have we learned in the past four decades? *Journal of Child Psychology and Psychiatry, 45*(1), 2-40.

Walker, M. P., Brakefield, T., Morgan, A., Hobson, J. A., & Stickgold, R. (2002). Practice with sleep makes perfect: Sleep-dependent motor skill learning. *Neuron, 35*(1), 205-211.

Walker, M. P., Stickgold, R., Alsop, D., Gaab, N., & Schlaug, G. (2005). Sleep-dependent motor memory plasticity in the human brain. *Neuroscience, 113*, 911-917.

Wechsler, D. (1993). *Wechsler objective reading dimension (word)*. London, UK: The Psychological Corporation.

Wechsler, D. (1998). *Wechsler adult intelligence scale - third edition*. London, UK: The Psychological Corporation.

Weible, A. P., Weiss, C., & Disterhoft, J. F. (2003). Activity profiles of single neurons in caudal anterior cingulate cortex during trace eyeblink conditioning in the rabbit. *Journal of Neurophysiology, 90*(2), 599-612.

Wimmer, H., Mayringer, H., & Raberger, T. (1999). Reading and dual-task balancing: Evidence against the automatization deficit explanation of developmental dyslexia. *Journal of Learning Disabilities, 32*(5), 473-478.

Witton, C., Talcott, J. B., Hansen, P. C., Richardson, A. J., Griffiths, T. D., Rees, A., et al. (1998). Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexic and normal readers. *Current Biology, 8*, 791-797.

Wolf, M., & Bowers, P. G. (1999). The double-deficit hypothesis for the developmental dyslexias. *Journal of Educational Psychology, 91*(3), 415-438.

World Federation of Neurology. (1968). *Report of research group on dyslexia and world illiteracy*. Dallas: WFN.

Wu, T., Kansaku, K., & Hallett, M. (2004). How self-initiated memorized movements become automatic: A functional mri study. *Journal of Neurophysiology, 91*(4), 1690-1698.

Yap, R. L., & van der Leij, A. (1993). Word-processing in dyslexics - an automatic decoding deficit. *Reading and Writing, 5*(3), 261-279.

Yap, R. L., & van der Leij, A. (1994). Testing the automatization deficit hypothesis of dyslexia via a dual-task paradigm. *Journal of Learning Disabilities, 27*(10), 660-665.

10. Appendices

10.1. Appendix 1: The adult dyslexia index

There are four aspects to the Adult Dyslexia Index (ADI) score each worth up to 1 point, thus the maximum ADI is 4. The first point is given according to previous (childhood/adolescent) diagnosis. The second 1.0 is for a spelling age of 15.6 years or less, (or 0.5 for < 17 years). The third is given for slow (0.5) or inaccurate (0.5) reading of a passage including nonsense words. The fourth is given for one (0.5) or more (1) impairments on the WAIS-III ACID subtests (Arithmetic, Digit-Symbol Coding, Information and Digit Span) relative to mean non-ACID performance.

10.2. Appendix 2: Example items from the Brown ADD scale (Brown, 1996).

Activation category: "Has a hard time waking up in the morning; finds it very difficult to get out of bed and to get going."

Attention category: ""Spaces out" involuntarily and frequently when doing required reading; keeps thinking of things that have nothing to do with what's being read."

Effort category: "Feels sleepy or tired during the day, even after a decent sleep the night before."

Affect category: "Appears apathetic or unmotivated (others think he/she doesn't care at all about his/her work)."

Memory category: "Often leaves out words or letters in writing."

10.3. Appendix 3: Study 1 interview (balance and postural control)

Here follows a summary of the interview conducted with each subject in study 1, each question is printed together with a description of the participants' responses.

10.3.1.1. Is English your first language? If no, for how long have you been a fluent English speaker?

100% answered that English was their first language.

10.3.1.2. Do you suffer from any orthopaedic or vestibular impairment?

78% answered "no", 22% (9) gave other answers. Of these, four were in the dyslexic group; one had sinusitis, one suffers from a recurring trapped nerve in his back but noted that it caused no trouble during the experiment, one had a history of MRI scans for childhood knee complaints but had not been seen for 2-3 years, another recalled a few old rugby related injuries. This leaves five from the control group; one reported being prone to hay fever and sinusitis but fine on the day of testing, a second mentioned being prone to sinusitis but untroubled that day, another complained of blocked sinuses and a twisted pelvis (which caused a little pain but no mobility problems), another reported having knee surgery 5 years previously with no enduring day to day effects and the fifth recalled osgoodschlatter on the knee (5-6 years earlier) again with no lasting effect.

10.3.1.3. Have you suffered any serious muscular or skeletal injury in the past year?

85% answered "no", of the remaining 6, four were in the dyslexic group and reported: (1) broken thumb (2) broken wrist 3 months earlier (3) "wrist problems" (4) dislocated shoulder ~ 12 months earlier. The remaining two participants from the control group reported: (1) torn ankle ligaments 6 months earlier and a full recovery since (2) pulled ankle ligaments with no lasting effect.

10.3.1.4. Are you expert in any of the following: Dance, Sport, Gymnastics?

For the purpose of this report, participation more than once per week was considered frequent. Some participants reported past engagement in relevant activities rather than current behaviour. They are represented separately in the table. Swimming, running, walking and going to the gym were all excluded from the below as they were considered not to constitute considerable practice in balance and motor coordination.

Numbers playing sports

	All participants		*Dyslexic*		*Control*	
	Number	% of tot	Number	% of tot	Number	% of tot
Currently Frequent	13	32.5%	8	40.0%	5	25.0%
Currently Infrequent	5	12.5%	1	5.0%	4	20.0%
Previously Frequent	5	12.5%	2	10.0%	3	15.0%
Previously Infrequent	1	2.5%	1	5.0%	0	0.0%
None	16	40.0%	8	40.0%	8	40.0%
Total	40	100.0%	20	100.0%	20	100.0%

Crucially, the number of people never participating is the same in each group (8) and the number participating often is similar, with the dyslexic group apparently more active. This strengthens any findings of poor motor control in the dyslexic group as their lifestyles should make them more controlled rather than less.

10.3.1.5. Are you suffering from any cold or congestion at present?

Colds and congestion

	All participants		*Dyslexic*		*Control*	
	Number	% of tot	Number	% of tot	Number	% of tot
None	30	75.0%	14	70.0%	16	80.0%
Mild cold symptoms	5	12.5%	3	15.0%	2	10.0%
Hay fever	4	10.0%	2	10.0%	2	10.0%
Other complaint	1	2.5%	1	5.0%	0	0.0%
Total	40	100.0%	20	100.0%	20	100.0%

Again the groups appear well matched with the clear majority of participants having no complaint at all.

10.3.1.6. Are you taking any medication at present that might interfere with your co-ordination or balance?

Three dyslexic participants and six controls were taking antihistamines at the time of the experiments. Of these, one dyslexic was also taking antibiotics and eye drops for sinusitis. One of the controls was also taking these eye drops and paracetamol. A second of these six controls was using Vicks cold medication. One other member of the dyslexic group was taking antibiotics, a second was using antidepressants and a third was diabetic.

10.3.1.7. Have you had an ear infection in the last 6 months?

Rine *et al.* (1998) excluded participants from their study if they had had an ear infection in the last 6 months. In this study, 35/40 (87.5%) of participants simply

answered no to the above question. The remaining five responses are as follows: (i) No, but did have mumps [D], (ii) no, but did have hearing problems as a toddler (wax related) [D],(iii) ongoing sinus problems [C], (iv) congestion 2 weeks earlier [C], (v) ear infection 7 months ago [D]. (A letter in square brackets indicates the participant's group).

10.3.1.8. Have you received any supplementary literacy support at school/university or privately?

Nine members of the dyslexic group had received no supplementary literacy support. Those who had were asked approximately how many years they had received support for. Eleven had received support, for five this had been over a period of more than 3 years. One of the controls reported having had such help at school, this seems odd, but there is no reason in the psychometric data to exclude him from the comparison group. Furthermore, this participant grew up outside the UK, and so the meaning of the question for him, and implications of his answer may not be the same as would be the case for the other participants.

10.3.1.9. Do you have any family members with learning difficulties?

Eight of the dyslexic group reported having family members with learning difficulties. One control group member has a sister with dyslexia, this participant scored lower on the digit span subtest than on his other IQ subtests but all his reading writing and spelling scores were at least in the normal range.

10.3.1.10. What (if anything) do you know about DDAT?

Thirty-nine of the forty participants were not aware of DDAT, one dyslexic participant recalled a feature on "Richard and Judy".

10.3.1.11. Alcohol in last 24 hours:

Eighteen of the dyslexic group and sixteen of the controls had consumed no alcohol in the 24 hours prior to testing. Two of the dyslexic group and four of the controls had consumed alcohol but none reported drinking more than 7 units. The drinking was always the evening before the day of testing.

10.4. Appendix 4: Study 2 interview (classical eyeblink conditioning)

There were far fewer questions posed in study 2. They are reported here with an outline of participants' responses.

10.4.1.1. Do you have any visual or hearing problems?

8/18 dyslexic and 7/16 controls gave responses other than "no" to this question. Of the dyslexics 4 are short-sighted, 1 long-sighted and a sixth wears glasses to read and has astigmatism. One reported good hearing having had hearing problems when young and the eighth reported a tendency to suffer from sinusitis. Of the controls, six are short-sighted (one also being colour blind) and one reported a sinus blockage.

10.4.1.2. Are you wearing contact lenses?

One participant wore contact lenses during the experiments but this seemed not to inhibit blinking (section 5.2.1.3.). A further two arrived wearing contact lenses but removed them for the experiment.

10.4.1.3. Are you right handed / left handed?

In the dyslexic group 14 out of 18 dyslexic participants reported right handedness, compared with 11 out of 16 in the control group.

10.5. Appendix 5: Study 3 interview (motor skill acquisition)

The questions asked in study 3 were posed in two stages, some before the beginning of the experiment and the remainder when the participants returned on the second day.

10.5.1.1. Do you have any history of mobility problems in your hands (if so which hand/both?)

5 dyslexic and 4 control participants did not answer "no". Of these, 1 participant with dyslexia reported that writing can cause swelling of her wrist and 1 control participant said that his hands occasionally become a little stiff. The remaining 7 participants had all had injuries in the past (from cuts to broken wrists) but none considered that they suffered significant enduring effects.

10.5.1.2. How much did you have to drink last night?

Most said that they had not consumed alcohol the night before the experiment. Two participants in each group had taken a moderate amount of alcohol (2 or 3 units) and one in each group had consumed a large amount of alcohol (more than 10 units).

10.5.1.3. Are you right handed / left handed?

Right-handedness was reported by 9/13 dyslexic and 10/12 control participants.

10.5.1.4. Do you play the piano/other musical instrument/professional typist etc.?

This question was included to provide a rough baseline of general manual dexterity. Answers were coded to produce a score from 1-4 according to the following rationale:

1 No particular finger sequencing skills.

2 Average amount of typing ***or*** substantial past evidence (e.g. played violin regularly as a child).

3 Above average amount of typing ***or*** other current skill (e.g. pianist) ***or*** both of the criteria for '2' above.

4 Both of the criteria from '3' above ***or*** one item from '3' above as well as some other evidence.

Using scores from this scheme, the average score was 2.46 (s.d. = 1.20) in the dyslexic group and 3.00 (s.d. = 1.35) in the control group. An independent groups t-test showed no difference between the groups ($t = 1.06$, n.s.).

The following questions were asked on day 2

10.5.1.5. How much did you have to drink last night?

Again, most participants had not consumed alcohol in the 24 hours between test sessions. Three dyslexic participants had had a large amount of alcohol (more than 10 units). Four control participants had consumed a more moderate amount (2-7 units).

10.5.1.6. Approximately how long did you sleep last night?

Most participants slept for between 6 and 10 hours between test sessions. One participant in each group reported 5 hours and a further control participant only slept for 3 hours. On average, dyslexic participants had more sleep between sessions (7.88 hours, s.d. = 1.21, compared with 6.71 hours, s.d. = 1.48), this difference was statistically significant ($t = 2.18$, $p < 0.05$). Furthermore, this strengthens the finding of impaired consolidation in the dyslexic group since sleep is thought to be conducive to consolidation (section 3.9).

10.5.1.7. Have you practiced the sequence at all?

Despite instruction on day 1 not to practice the learned sequence before returning on day 2, 4 dyslexic participants and 3 control participants admitted some limited repetition in the intervening period.

10.5.1.8. Have you played piano/typed etc in the last 24 hours?

Minimal typing was performed by 4 dyslexic participants and one control, while 3 controls and one dyslexic did a more substantial amount of typing. A further control participant played the guitar between tests.

10.6. Appendix 6: Data tables

Appendix 6a: Participants in study 1: Balance and Postural Control

	Participant	D1	D2	D3	D4	D5	D6	D7	D8	D9
	Age	19	19	19	25	21	20	20	21	20
	Sex	M	F	M	M	M	M	F	M	M
	Height (cm)	176	170	175	180	175	187	179	187	190
	IQ (short-form WAIS-III)	151	110	133	(110)	121	130	113	108	126
WAIS-III subtest age-scaled scores	Vocabulary	17	11	14	•	12	11	12	10	11
	Similarities	17	10	14	•	13	12	13	10	15
	Digit Span	9	9	9	•	8	8	11	8	10
	Picture Completion	15	9	15	•	13	15	12	12	13
	Digit-Symbol Coding	8	9	10	•	10	10	8	7	9
	Block Design	16	16	14	•	14	18	11	13	15
	Brown ADD score	69	39	53	88	45	55	52	48	98
	NWR time (s)	67	61	88	•	171	76	65	77	90
	NWR errors made	18	9	16	•	19	13	8	10	15
	WORD spelling (raw score)	35	41	34	•	36	37	46	40	35
	DAST 1-minute reading	81	89	82	•	33	74	103	74	65
	DAST 1-minute writing	23	30	29	•	26	46	19	28	30
	DAST 2-minute spelling	19	31	29	•	27	28	27	35	28
Sensory Organisation Test (SOT)	C1	88	82	96	95	96	95	95	97	94
	C2	74	84	92	94	92	93	94	96	94
	C3	90	92	94	93	95	94	94	96	94
	C4	84	82	89	62	97	94	87	92	92
	C5	65	59	58	65	81	71	51	75	71
	C6	75	68	74	5	89	91	81	75	81
	C1 + Counting	92	81	91	72	94	94	92	95	93
	C1 + Slow CRT	92	89	88	84	95	94	92	96	96
	C1 + Fast CRT	89	86	93	86	96	92	94	96	96

D10	D11	D12	D13	D14	D15	D16	D17	D18	D19	D20	Mean	St. Dev.
21	20	20	18	20	34	20	41	20	18	20	**21.80**	*5.67*
M	M	F	F	F	M	M	M	M	M	F		
176	192	166	171	169	172	182	176	181	183	173	**178.00**	*7.20*
140	111	115	101	103	123	130	111	128	111	117	**120.11**	*13.04*
14	13	12	10	12	14	13	13	13	11	11	**12.32**	*1.70*
17	13	15	9	12	11	18	9	15	13	14	**13.16**	*2.65*
15	9	7	7	7	7	10	7	10	9	10	**8.95**	*1.93*
13	7	12	8	12	15	13	14	11	11	12	**12.21**	*2.30*
9	6	8	7	10	7	8	5	10	9	9	**8.37**	*1.46*
16	14	10	14	6	13	12	11	16	12	13	**13.37**	*2.73*
51	64	42	48	46	49	20	45	61	48	41	**53.10**	*17.04*
98	71	86	144	58	60	67	83	52	121	63	**84.11**	*30.96*
10	11	11	10	17	8	9	8	13	6	13	**11.79**	*3.75*
38	43	39	36	38	41	40	38	41	38	39	**38.68**	*2.98*
108	77	84	33	109	80	95	92	102	80	91	**81.68**	*20.95*
29	28	33	30	34	29	31	24	32	29	33	**29.63**	*5.41*
27	30	32	25	29	29	32	28	33	29	25	**28.58**	*3.49*
94	92	98	97	96	95	92	94	95	96	92	**93.95**	*3.61*
•	88	94	96	96	89	93	94	90	92	91	**91.37**	*5.17*
•	86	97	95	97	96	90	96	96	97	92	**93.89**	*2.88*
•	82	90	92	80	73	80	87	78	88	84	**84.89**	*8.24*
•	61	73	76	42	48	52	41	61	65	63	**62.00**	*11.36*
•	67	46	80	29	45	49	43	70	79	64	**63.74**	*22.20*
94	87	95	93	93	95	92	92	79	91	90	**90.25**	*6.08*
96	90	94	96	94	92	88	93	91	95	92	**92.35**	*3.27*
97	92	96	96	96	94	94	95	95	96	95	**93.70**	*3.25*

St. Err.	n	C1	C2	C3	C4	C5	C6	C7	C8	C9	C10	C11
1.27	20	22	22	22	21	23	20	22	19	22	23	19
		M	F	M	M	M	M	M	F	F	F	F
1.61	20	178	169	186	185	176	179	186	173	179	161	158
2.99	19	130	130	98	113	111	106	128	145	110	140	108
0.39	19	15	15	13	18	14	15	15	16	12	14	17
0.61	19	14	14	8	11	8	10	14	14	10	13	12
0.44	19	13	9	16	11	12	12	18	13	8	10	12
0.53	19	12	12	8	8	12	8	13	15	9	15	7
0.34	19	12	13	14	14	11	11	12	13	14	13	7
0.63	19	15	15	10	11	13	11	13	17	15	18	9
3.81	20	53	54	42	57	47	15	38	33	14	29	38
7.10	19	50	50	34	44	74	48	31	35	56	36	48
0.86	19	6	6	2	7	7	4	4	6	6	7	1
0.68	19	46	47	49	45	45	44	47	44	44	48	44
4.81	19	133	102	137	124	98	109	125	122	111	111	94
1.24	19	34	39	41	40	39	43	36	37	32	35	33
0.80	19	40	38	35	35	35	38	38	34	31	38	31
0.81	20	96	96	97	96	97	94	96	94	94	94	96
1.19	19	92	92	94	94	95	93	96	90	96	90	94
0.66	19	94	97	95	95	97	96	96	95	97	92	95
1.89	19	91	86	95	92	89	92	92	95	86	87	80
2.61	19	55	53	81	63	64	70	77	67	63	72	61
5.09	19	54	75	85	74	55	64	72	79	52	76	30
1.36	20	94	96	94	97	90	94	96	90	94	90	94
0.73	20	96	96	95	94	89	97	96	93	96	94	94
0.73	20	96	96	94	96	89	97	97	94	96	94	93

C12	C13	C14	C15	C16	C17	C18	C19	C20	Mean	St. Dev.	St. Err.	n
21	20	24	22	22	19	22	23	19	**21.35**	*1.53*	0.34	20
M	M	M	F	M	M	M	M	F				
183	182	174	165	187	183	169	172	154	**174.95**	*9.78*	2.19	20
133	111	145	99	137	119	111	145	101	**121.00**	*16.22*	3.63	20
19	14	18	11	18	12	18	17	12	**15.15**	*2.41*	0.54	20
14	13	15	11	12	11	13	15	10	**12.10**	*2.15*	0.48	20
14	12	15	11	15	8	10	17	9	**12.25**	*2.90*	0.65	20
12	9	12	8	13	12	5	15	8	**10.65**	*2.94*	0.66	20
15	11	17	15	14	11	9	16	8	**12.50**	*2.59*	0.58	20
12	11	17	10	16	16	11	15	11	**13.30**	*2.75*	0.62	20
31	15	44	50	27	17	42	32	14	**34.60**	*14.27*	3.19	20
39	43	38	35	49	47	50	36	42	**44.25**	*9.85*	2.20	20
5	4	3	8	4	6	2	1	4	**4.65**	*2.08*	0.47	20
45	47	47	44	43	44	48	46	48	**45.75**	*1.77*	0.40	20
135	119	126	120	123	111	122	125	119	**118.30**	*11.68*	2.61	20
31	33	40	35	41	34	32	34	33	**36.10**	*3.61*	0.81	20
34	37	39	36	31	34	39	36	40	**35.95**	*2.87*	0.64	20
96	96	96	96	95	96	95	96	93	**95.43**	*1.12*	0.25	20
93	96	94	97	91	93	93	96	91	**93.50**	*2.09*	0.47	20
95	98	97	96	85	97	94	96	90	**94.85**	*2.98*	0.67	20
92	92	91	92	76	93	77	91	82	**88.55**	*5.69*	1.27	20
71	71	68	62	69	67	56	72	64	**66.30**	*7.09*	1.58	20
81	73	34	60	66	67	70	45	76	**64.40**	*15.24*	3.41	20
93	93	93	92	72	92	90	96	88	**91.90**	*5.27*	1.18	20
96	97	97	94	89	96	93	96	92	**94.50**	*2.37*	0.53	20
94	97	97	96	96	95	94	96	94	**95.05**	*1.90*	0.43	20

Adaptation Test (ADT) alone	Toes up 1	101	62	89	84	72	82	91	109	126
	Toes up 2	88	51	68	80	73	65	81	104	89
	Toes up 3	77	42	66	•	62	61	88	99	87
	Toes up 4	86	58	84	85	66	84	89	•	79
	Toes up 5	79	47	67	78	54	64	111	101	100
	Toes up gradient	-4.6	-2.3	-2.8	-0.7	-4.3	-1.7	4.8	-1.9	-6.2
	Toes up interc't	100	59	83	84	78	76	78	109	115
	Toes down 1	80	65	53	67	62	49	65	74	76
	Toes down 2	72	40	39	18	56	•	56	•	•
	Toes down 3	85	37	27	6	41	40	48	70	62
	Toes down 4	82	44	49	10	37	38	46	75	55
	Toes down 5	80	30	32	49	61	37	50	80	54
	Toes down gradient	1.0	-6.6	-3.2	-4.4	-2.1	-3.1	-4.0	1.4	-5.8
	Toes down intercept	77	63	50	43	58	51	65	70	81
ADT + Counting	Toes up 1	82	69	72	•	93	68	71	102	83
	Toes up 2	76	34	82	•	61	66	73	65	•
	Toes up 3	89	37	62	•	55	48	•	60	69
	Toes up 4	•	26	66	•	50	56	65	77	71
	Toes up 5	83	29	51	•	62	38	59	73	69
	Toes up gradient	1.0	-8.8	-5.8	•	-7.3	-7.0	-3.2	-4.6	-3.4
	Toes up interc't	80	65	84	•	86	76	77	89	84
	Toes down 1	64	45	52	•	46	26	52	62	•
	Toes down 2	52	61	42	•	47	•	•	72	•
	Toes down 3	•	35	45	•	40	36	48	41	•
	Toes down 4	53	52	35	•	35	39	39	52	•
	Toes down 5	52	26	34	•	39	39	36	42	•
	Toes down gradient	-2.3	-4.7	-4.3	•	-2.6	3.4	-4.2	-6.0	•
	Toes down intercept	62	58	55	•	49	24	57	72	•
ADT + Slow CRT	Toes up 1	85	67	56	54	71	75	58	113	68
	Toes up 2	•	45	•	74	76	76	62	•	69
	Toes up 3	90	65	52	66	65	65	62	93	•
	Toes up 4	93	34	49	63	71	71	74	86	80
	Toes up 5	92	49	51	40	59	53	70	90	74
	Toes up gradient	1.9	-4.7	-1.5	-3.9	-2.9	-4.9	3.6	-6.3	2.3
	Toes up interc't	84	66	57	71	77	83	54	116	66
	Toes down 1	63	37	41	50	48	54	58	75	66
	Toes down 2	85	27	41	47	42	45	43	66	44
	Toes down 3	64	23	31	•	26	27	41	•	48
	Toes down 4	70	29	30	41	26	26	51	55	58
	Toes down 5	56	28	31	34	38	29	42	52	49

84	61	150	92	150	98	150	103	91	119	90	100.20	26.82
•	64	72	85	57	72	98	145	105	134	89	85.26	24.23
73	76	80	•	58	95	78	146	80	133	•	82.41	25.80
65	78	79	71	69	63	88	87	101	116	85	80.68	13.83
60	56	86	67	71	65	89	92	76	114	78	77.75	18.90
-6.1	0.4	-12.1	-6.4	-14.6	-7.5	-13.2	-8.0	-3.4	-2.8	-2.8	-4.81	4.69
90	66	130	98	125	101	140	139	101	132	94	99.81	24.02
64	69	81	64	57	54	69	110	74	76	66	68.75	13.10
51	45	85	66	38	59	47	108	58	68	61	56.88	20.33
•	33	55	56	23	56	37	89	44	63	24	47.16	21.24
52	34	46	57	27	52	33	86	29	•	64	48.21	19.23
42	33	41	49	26	59	40	97	33	38	23	47.70	19.59
-4.3	-8.3	-11.9	-3.9	-7.3	0.3	-7.2	-4.8	-11.1	-9.5	-8.3	-5.15	3.72
65	68	97	70	56	55	67	112	81	87	73	69.42	16.69
49	82	94	93	56	62	77	150	93	100	79	82.89	21.89
46	75	83	89	54	78	62	•	76	61	84	68.53	14.57
46	71	76	77	72	77	60	126	•	69	73	68.65	19.77
45	64	65	79	59	83	59	99	59	73	78	65.22	16.13
60	53	77	76	64	71	64	88	52	47	73	62.58	15.12
2.1	-6.9	-5.2	-4.4	2.1	2.3	-2.9	-16.1	-9.9	-9.4	-1.8	-4.70	4.73
43	90	95	96	55	67	73	168	100	98	83	84.66	25.00
60	47	52	69	50	60	•	76	71	46	49	54.53	12.05
42	50	56	51	•	59	•	74	71	63	34	55.29	12.14
36	44	55	53	41	58	•	97	73	65	39	50.38	16.58
28	•	54	39	34	50	•	96	60	52	30	46.75	16.41
36	49	64	48	32	50	•	75	59	39	29	44.06	13.06
-6.2	0.2	2.2	-5.4	-4.7	-2.9	•	2.0	-3.5	-2.5	-4.4	-2.70	2.95
59	47	50	68	55	64	•	78	77	61	49	57.89	12.86
64	64	83	70	50	66	60	103	77	96	64	72.20	16.57
65	63	107	73	45	74	63	112	91	77	77	73.47	17.71
66	63	79	72	66	73	61	•	82	70	72	70.11	10.32
68	•	89	•	61	75	66	121	105	86	78	76.11	19.78
48	54	77	70	43	70	74	113	76	73	62	66.90	18.23
-2.9	-2.5	-3.0	-0.2	0.2	0.9	3.1	2.9	1.2	-3.7	-0.3	-1.04	3.00
71	68	96	72	52	69	56	104	83	92	72	75.32	16.80
47	45	60	67	52	55	58	82	54	58	64	56.70	11.01
42	•	•	43	27	59	53	74	31	77	30	48.67	17.22
33	34	42	46	30	58	50	87	33	45	24	41.22	16.34
28	36	39	53	25	51	38	90	37	63	26	43.60	17.53
34	29	45	45	29	52	31	71	27	45	23	39.50	12.28

6.00	20	89	63	80	86	80	98	113	81	82	88	65
5.56	19	108	64	49	91	66	80	83	69	69	88	70
6.26	17	98	63	72	91	53	•	77	70	68	81	78
3.17	19	78	•	44	•	64	83	90	64	66	•	60
4.23	20	79	61	50	73	73	72	87	71	76	78	69
1.05	20	-5.0	-0.6	-6.5	-3.6	-1.6	-4.9	-4.5	-2.5	-1.5	-2.8	-0.2
5.37	20	105	64	79	95	72	98	104	79	77	91	69
2.93	20	52	52	55	58	49	56	83	34	70	85	56
4.93	17	35	45	38	64	45	•	51		52	43	41
4.87	19	33	•	41	59	35	43	36	33	43	26	45
4.41	19	30	30	30	58	31	40	25	29	40	28	27
4.38	20	31	34	59	60	32	38	48	29	28	28	25
0.83	20	-4.7	-5.1	0.0	-0.2	-4.8	-4.6	-9.6	-1.4	-9.6	-12.9	-7.6
3.73	20	50	56	45	60	53	59	77	36	75	81	62
5.02	19	76	74	73	104	45	122	122	87	73	83	64
3.53	17	66	68	45	117	54	98	103	63	78	79	66
4.80	17	•	73	54	108	57	87	95	56	72	78	63
3.80	18	57	74	62	68	•	68	86	52	80	83	61
3.47	19	58	70	44	91	49	70	84	45	63	77	63
1.09	19	-4.5	-0.2	-4.1	-7.5	0.6	-13.4	-9.3	-9.5	-1.8	-0.8	-0.7
5.73	19	78	72	68	120	50	129	126	89	79	82	66
2.92	17	70	63	45	•	43	71	74	42	45	60	48
3.24	14	54	60	46	•	39	48	45	•	31	65	•
4.15	16	34	•	36	•		61	63	34	26	47	34
4.10	16	25	39	28	•	34	58	45	33	37	42	24
3.17	17	31	38	37	•	26	40	46	29	43	42	21
0.71	17	-10.7	-7.1	-3.4	•	-3.9	-5.2	-5.6	-3.1	0.2	-5.9	-7.1
3.12	17	75	71	49	•	47	71	71	45	36	69	55
3.71	20	86	76	51	96	51	103	120	89	58	55	72
4.30	17	56	76	69	87	47	70	103	52	67	•	84
2.43	18	54	76	51	73	50	71	•	52	58	56	63
4.66	18	53	77	49	64	•	81	86	53	61	62	73
4.08	20	73	77	65	73	47	70	77	48	74	63	68
0.67	20	-2.9	0.3	0.8	-6.9	-0.7	-5.5	-10.3	-8.1	2.6	2.2	-1.9
3.76	20	73	76	55	99	51	96	127	83	56	52	78
2.46	20	55	66	57	54	42	44	86	30	71	57	59
4.06	18	48	52	41	•	28	42	48	39	50	43	67
3.85	18	•	50	55	54	27	38	34	25	40	42	31
3.92	20	46	40	48	57	20	49	48	33	24	38	29
2.74	20	48	28	58	51	19	42	47	20	36	42	33

85	87	139	77	71	112	72	95	52	**85.75**	*19.46*	4.35	20
100	75	113	83	59	86	81	108	64	**80.30**	*17.43*	3.90	20
91	60	114	63	•	92	92	68	•	**78.29**	*16.07*	3.90	17
62	64	116	64	58	70	82	•	63	**70.50**	*16.50*	4.12	16
72	74	81	69	67	69	72	83	64	**72.00**	*8.13*	1.82	20
-6.4	-3.7	-11.3	-3.5	-0.9	-10.2	0.1	-5.0	2.3	**-3.61**	*3.34*	0.75	20
101	83	147	82	66	116	80	102	54	**88.17**	*21.22*	4.75	20
80	49	80	69	44	90	98	82	56	**64.90**	*17.56*	3.93	20
45	48	•	72	•	70	65	•	37	**50.07**	*12.12*	3.13	15
32	•	56	51	37	66	44	50	23	**41.83**	*11.40*	2.69	18
42	42	50	52	32	51	45	43	25	**37.50**	*10.09*	2.26	20
42	40	47	42	32	54	56	29	31	**39.25**	*11.26*	2.52	20
-7.9	-2.4	-8.5	-7.4	-3.2	-9.1	-10.4	-13.0	-6.2	**-6.43**	*3.84*	0.86	20
72	52	86	79	47	94	93	93	53	**66.11**	*17.77*	3.97	20
76	142	124	82	83	150	88	102	60	**91.50**	*27.91*	6.24	20
76	59	96	67	66	•	84	102	57	**76.00**	*19.37*	4.44	19
•	60	87	81	65	71	70	107	55	**74.39**	*16.96*	4.00	18
66	49	72	79	63	62	•	•	50	**66.59**	*11.30*	2.74	17
67	59	81	89	50	61	67	93	42	**66.15**	*15.88*	3.55	20
-2.8	-17.6	-11.0	2.6	-6.9	-23.1	-5.6	-2.2	-4.3	**-6.10**	*6.45*	1.44	20
80	127	125	72	86	161	93	107	66	**93.69**	*28.82*	6.44	20
56	55	54	80	56	74	77	63	60	**59.79**	*12.06*	2.77	19
51	38	•	61	58	53	44	56	67	**51.00**	*10.17*	2.54	16
46	41	53	60	53	43	42	•	67	**46.25**	*12.20*	3.05	16
•	38	43	43	•	44	43	40	46	**38.94**	*8.43*	2.04	17
37	•	35	40	30	43	49	38	49	**37.44**	*7.72*	1.82	18
-4.7	-4.8	-4.7	-9.8	-6.9	-7.1	-5.7	-6.6	-4.3	**-5.61**	*2.41*	0.55	19
61	55	62	86	68	73	68	69	71	**63.21**	*12.56*	2.88	19
45	80	114	67	59	104	84	104	47	**78.05**	*23.56*	5.27	20
73	71	104	71	56	64	66	•	43	**69.94**	*16.83*	3.97	18
77	50	94	74	•	60	77	84	49	**64.94**	*13.60*	3.20	18
46	44	83	•	63	76	70	84	•	**66.18**	*13.89*	3.37	17
57	71	104	71	61	57	81	84	38	**67.95**	*14.61*	3.27	20
-0.3	-4.5	-4.1	0.9	1.1	-8.2	-0.2	-5.1	-1.9	**-2.64**	*3.78*	0.85	20
61	77	112	68	56	97	76	106	50	**77.35**	*22.57*	5.05	20
57	46	65	75	58	56	72	63	44	**57.85**	*12.95*	2.89	20
39	43	59	80	34	58	57	55	29	**48.00**	*12.93*	2.97	19
34	42	45	46	•	51	39	46	30	**40.50**	*9.06*	2.13	18
34	39	39	41	35	48	49	43	33	**39.65**	*9.26*	2.07	20
30	33	39	37	52	40	60	52	33	**40.00**	*11.50*	2.57	20

	Toes down gradient	-2.9	-1.6	-3.1	-3.8	-3.6	-6.9	-2.4	-5.7	-2.0
	Toes down intercept	76	34	44	54	47	57	54	79	59
ADT + Fast CRT	Toes up 1	71	35	59	61	57	59	63	92	90
	Toes up 2	71	•	62	62	64	68	65	87	72
	Toes up 3	84	39	66	60	65	53	67	70	66
	Toes up 4	82	50	64	62	•	52	67	68	58
	Toes up 5	101	46	49	55	68	66	90	85	71
	Toes up gradient	7.1	3.4	-1.8	-1.2	2.5	-0.2	5.6	-3.3	-5.2
	Toes up interc't	61	32	65	64	57	60	54	90	87
	Toes down 1	44	33	35	56	71	44	55	79	54
	Toes down 2	49	21	24	56	•	•	45	72	56
	Toes down 3	43	35	28	45	52	44	41	47	•
	Toes down 4	40	23	28	42	51	33	42	44	55
	Toes down 5	34	61	30	42	44	36	31	43	52
	Toes down gradient	-2.9	5.8	-0.6	-4.2	-6.6	-2.5	-5.1	-10.0	-0.5
	Toes down intercept	51	17	31	61	76	48	58	87	56
Heel-to-toe alone	x-axis	•	1.43	7.74	2.24	1.11	•	1.90	0.91	0.56
	y-axis	•	0.83	10.16	0.95	1.55	•	1.89	1.22	0.70
	z-axis	•	1.00	4.29	1.04	0.86	•	1.03	0.56	0.40
	xyz	•	1.09	7.40	1.41	1.17	•	1.60	0.90	0.55
Heel-to-toe + Blindfold	x-axis	•	1.66	3.71	4.04	0.95	•	3.00	0.95	0.72
	y-axis	•	0.47	4.96	1.33	1.71	•	3.14	1.59	0.85
	z-axis	•	0.72	1.88	1.86	0.92	•	1.22	0.64	0.51
	xyz	•	0.95	3.51	2.41	1.19	•	2.45	1.06	0.69
Heel-to-toe + Counting	x-axis	•	6.88	3.64	3.10	•	•	•	0.59	3.79
	y-axis	•	2.20	3.16	1.53	•	•	•	1.05	4.14
	z-axis	•	4.85	1.13	1.92	0.78	•	•	0.43	1.90
	xyz	•	4.64	2.64	2.18	•	•	•	0.69	3.28
Heel-to-toe + Slow CRT	x-axis	•	7.35	1.82	1.36	3.01	•	1.32	0.58	1.14
	y-axis	•	1.97	2.39	1.92	3.00	•	2.06	1.59	1.64
	z-axis	•	5.76	0.95	1.14	1.66	•	0.61	0.60	1.23
	xyz	•	5.03	1.72	1.47	2.56	•	1.33	0.93	1.33
Heel-to-toe + Fast CRT	x-axis	•	4.60	2.97	2.43	6.00	•	1.39	0.85	2.10
	y-axis	•	1.04	2.53	4.31	4.32	•	1.73	2.05	2.48
	z-axis	•	2.28	1.10	1.80	2.57	•	0.79	0.65	1.68
	xyz	•	2.64	2.20	2.85	4.30	•	1.30	1.18	2.08
Counting (% correct)	During SOT	96	79	100	67	88	92	100	100	88
	During ADT	89	59	76	•	81	•	84	95	92
	During heel-to-toe	•	68	68	•	71	•	97	97	•
Slow CRT (% correct)	During SOT	100	100	100	100	100	100	100	100	100
	During ADT	82	100	91	100	91	91	91	91	92
	During heel-to-toe	100	100	100	100	100	91	100	100	100
Fast CRT (% correct)	During SOT	100	•	100	89	100	100	100	89	•
	During ADT	96	89	96	89	100	100	100	85	92
	During heel-to-toe	100	100	100	94	100	100	100	100	•

-4.0	-3.7	-4.3	-3.4	-4.8	-1.4	-6.9	-0.6	-4.8	-4.0	-8.6	**-3.92**	*1.99*
49	48	60	61	47	59	67	83	51	70	59	**57.88**	*12.39*
65	64	77	82	40	67	82	97	84	71	64	**69.00**	*16.06*
62	65	82	79	61	65	72	87	61	63	63	**69.00**	*8.69*
66	•	63	69	53	•	78	89	63	55	59	**64.72**	*11.60*
•	58	65	•	67	63	89	118	60	47	•	**66.88**	*17.30*
62	55	65	59	63	62	77	95	65	75	53	**68.10**	*15.11*
-0.5	-2.5	-4.1	-6.0	5.2	-1.2	0.7	2.7	-3.9	-0.8	-2.9	**-0.32**	*3.70*
65	68	83	89	41	68	78	89	78	65	68	**67.99**	*15.72*
81	60	49	53	56	53	70	87	50	52	46	**56.40**	*14.57*
27	47	45	46	43	•	50	84	48	33	33	**45.82**	*16.14*
33	33	54	•	39	50	38	70	41	54	23	**42.78**	*11.01*
31	31	37	37	25	49	36	•	39	52	18	**37.53**	*10.18*
34	49	47	31	26	48	52	61	27	41	20	**40.45**	*11.48*
-9.0	-3.8	-1.2	-5.3	-7.8	-1.3	-5.0	-6.9	-5.5	-0.3	-6.7	**-3.97**	*3.67*
68	55	50	58	61	54	64	95	58	47	48	**57.09**	*17.03*
2.56	2.31	0.60	0.83	3.10	0.97	3.97	0.72	3.00	1.49	•	**2.08**	*1.77*
3.16	1.29	1.70	0.99	2.15	0.59	5.46	1.17	2.33	1.40	•	**2.21**	*2.36*
1.02	1.20	0.77	0.37	1.20	0.43	0.98	0.65	1.80	1.02	•	**1.09**	*0.90*
2.25	1.60	1.02	0.73	2.15	0.66	3.47	0.84	2.38	1.31	•	**1.80**	*1.63*
2.11	2.78	1.55	1.05	2.64	1.04	5.98	0.82	1.98	6.92	•	**2.46**	*1.82*
2.67	1.61	1.79	1.29	1.32	0.61	7.98	1.87	1.96	5.82	•	**2.41**	*2.02*
1.38	1.39	1.08	0.42	1.07	0.62	2.01	0.85	1.04	1.80	•	**1.14**	*0.51*
2.05	1.93	1.47	0.92	1.68	0.75	5.32	1.18	1.66	4.85	•	**2.00**	*1.37*
1.83	2.98	0.33	0.46	3.65	1.11	5.60	0.85	2.49	1.96	•	**2.62**	*1.92*
1.32	1.47	1.54	0.72	5.71	1.20	5.19	1.61	3.05	2.28	•	**2.41**	*1.54*
0.98	2.13	0.56	0.33	1.21	0.62	1.27	1.07	0.92	0.76	•	**1.30**	*1.09*
1.38	2.19	0.81	0.50	3.52	0.98	4.02	1.18	2.15	1.67	•	**2.12**	*1.28*
2.28	2.16	0.39	0.67	6.32	1.10	2.07	0.94	2.21	1.41	•	**2.12**	*1.91*
1.67	1.38	1.06	0.89	5.64	1.47	5.00	0.60	1.74	1.68	•	**2.10**	*1.34*
0.91	1.64	0.41	0.30	1.06	0.88	1.84	0.71	1.38	0.57	•	**1.27**	*1.24*
1.62	1.73	0.62	0.62	4.34	1.15	2.97	0.75	1.78	1.22	•	**1.83**	*1.25*
2.19	1.97	0.48	0.91	1.35	1.17	4.90	0.97	2.92	1.73	•	**2.29**	*1.56*
1.38	1.24	1.02	0.87	0.97	1.84	5.38	0.63	1.22	2.24	•	**2.07**	*1.38*
0.90	1.43	0.54	0.40	0.82	0.87	2.49	0.78	2.06	0.69	•	**1.28**	*0.72*
1.49	1.55	0.68	0.72	1.05	1.29	4.26	0.79	2.06	1.55	•	**1.88**	*1.10*
100	72	•	68	68	100	88	83	59	49	75	**82.74**	*15.65*
100	80	•	95	67	97	65	86	81	67	70	**81.41**	*12.51*
78	71	93	90	•	97	87	80	93	97	84	**84.73**	*11.27*
100	100	88	100	•	100	100	100	100	100	88	**98.74**	*3.78*
100	91	91	91	91	91	91	92	100	91	100	**92.90**	*4.68*
100	91	100	82	91	100	100	100	100	100	100	**97.75**	*4.95*
100	78	83	•	94	100	100	100	100	100	100	**96.06**	*7.01*
100	67	74	•	96	88	96	97	100	100	100	**92.89**	*9.29*
100	88	94	59	•	100	100	100	100	100	100	**96.39**	*9.89*

0.45	20	-1.6	-8.8	0.9	-0.3	-5.4	0.3	-7.8	-2.6	-9.6	-3.5	-9.0
2.77	20	54	74	49	55	43	42	76	37	73	55	71
3.59	20	62	59	44	71	49	79	92	51	50	79	75
1.99	19	63	69	38	73	52	68	70	•	50	65	71
2.73	18	•	57	46	72	•	63	76	51	•	60	•
4.33	16	58	65	54	65	51	62	67	54	65	57	64
3.38	20	58	57	51	69	56	71	70	52	62	69	62
0.83	20	-1.3	-0.8	3.0	-1.2	1.3	-2.2	-4.7	0.5	3.9	-2.8	-3.3
3.51	20	64	64	38	74	48	75	89	51	45	74	78
3.26	20	49	67	61	53	46	50	64	37	83	65	49
3.91	17	37	50	49	69	28	49	50	•	43	46	40
2.59	18	31	32	•	68	29	52	46	28	43	45	•
2.34	19	•	31	37	63	24	48	35	29	50	48	32
2.57	20	27	29	36	64	19	49	44	23	51	34	26
0.82	20	-5.1	-9.5	-6.2	1.6	-5.8	-0.3	-5.5	-3.2	-5.7	-6.0	-5.4
3.81	20	50	70	64	59	47	51	64	40	71	66	53
0.43	17	2.38	0.40	3.56	3.38	1.17	1.27	2.71	0.57	1.08	0.84	0.42
0.57	17	1.17	0.99	2.88	2.24	1.61	1.49	2.01	0.98	1.46	0.86	0.96
0.22	17	1.04	0.39	1.81	1.99	0.54	0.61	1.04	0.35	0.54	0.45	0.49
0.40	17	1.53	0.59	2.75	2.54	1.11	1.13	1.92	0.63	1.03	0.72	0.62
0.44	17	2.63	0.94	3.56	3.41	1.15	1.43	3.10	0.96	3.59	0.99	0.56
0.49	17	1.39	1.40	3.70	2.76	1.48	1.26	2.96	1.43	6.19	1.14	0.65
0.12	17	1.00	0.54	1.84	2.15	0.47	0.58	1.62	0.49	2.11	0.48	0.64
0.33	17	1.67	0.96	3.03	2.77	1.03	1.09	2.56	0.96	3.96	0.87	0.62
0.49	15	1.63	0.53	1.44	2.96	1.69	0.94	2.12	0.57	0.64	0.96	0.44
0.40	15	0.93	1.38	1.95	1.99	1.97	1.34	1.74	1.09	1.16	0.99	0.83
0.27	16	0.83	0.47	0.93	2.14	0.75	0.48	1.09	0.50	0.38	0.43	0.72
0.33	15	1.13	0.79	1.44	2.36	1.47	0.92	1.65	0.72	0.73	0.79	0.67
0.46	17	1.51	0.39	2.56	3.17	1.55	0.67	1.98	0.59	0.80	0.70	0.40
0.32	17	1.72	0.97	2.84	1.53	1.87	0.91	1.30	1.17	1.37	0.93	1.13
0.30	17	0.72	0.41	0.99	2.53	0.72	0.41	1.28	0.40	0.41	0.46	0.50
0.30	17	1.32	0.59	2.13	2.41	1.38	0.66	1.52	0.72	0.86	0.70	0.68
0.38	17	3.08	0.45	2.12	3.93	2.99	0.72	1.64	0.44	0.97	0.79	0.56
0.33	17	1.31	0.98	3.21	2.13	2.40	0.88	1.43	0.88	1.81	1.02	1.21
0.17	17	0.98	0.36	1.50	2.82	1.04	0.40	0.88	0.36	0.36	0.49	0.84
0.27	17	1.79	0.60	2.28	2.96	2.14	0.67	1.32	0.56	1.04	0.76	0.87
3.59	19	81	100	88	100	100	100	50	60	83	•	100
3.03	17	85	100	86	100	97	100	71	94	81	•	100
2.91	15	94	100	100	100	100	100	61	97	93	•	100
0.87	19	100	100	88	100	100	100	100	100	100	•	100
1.05	20	100	91	100	91	91	100	91	100	100	100	91
1.11	20	100	100	100	100	100	100	100	100	100	100	100
1.70	17	100	100	100	100	100	100	100	100	100	•	100
2.13	19	96	96	100	100	100	100	96	96	100	•	96
2.33	18	100	100	100	100	100	100	100	100	100	100	100

-5.9	-3.0	-7.2	-11.5	-1.1	-4.2	-3.2	-3.4	-1.8	**-4.44**	*3.56*	0.80	20
57	50	71	90	48	63	65	62	39	**58.71**	*14.12*	3.16	20
48	76	67	69	46	56	81	93	41	**64.40**	*16.06*	3.59	20
55	91	80	70	72	68	•	132	46	**68.50**	*20.28*	4.78	18
50	90	75	74	68	•	69	76	46	**64.87**	*12.98*	3.35	15
63	81	69	•	47	54	72	77	47	**61.68**	*9.41*	2.16	19
44	72	81	78	71	48	64	102	34	**63.55**	*14.87*	3.32	20
0.0	-1.8	1.7	2.4	2.5	-3.0	-3.8	-3.7	-1.3	**-0.73**	*2.53*	0.57	20
52	87	69	66	53	66	84	107	47	**66.54**	*17.63*	3.94	20
63	46	60	60	51	54	60	61	46	**56.25**	*10.19*	2.28	20
38	39	48	47	51	43	46	50	32	**45.00**	*8.74*	2.00	19
30	•	50	50	34	41	40	52	25	**40.94**	*11.45*	2.78	17
28	47	39	48	53	41	37	48	30	**40.42**	*10.30*	2.36	19
30	41	36	39	36	37	27	50	33	**36.55**	*10.97*	2.45	20
-7.6	-0.2	-5.7	-4.1	-2.8	-3.6	-7.5	-2.4	-2.8	**-4.39**	*2.73*	0.61	20
61	44	64	61	53	54	65	59	42	**56.82**	*9.29*	2.08	20
1.04	1.58	1.78	0.48	3.99	0.79	1.98	2.93	0.52	**1.64**	*1.14*	0.26	20
1.05	1.68	1.43	1.10	4.96	1.42	3.33	1.51	1.73	**1.74**	*0.99*	0.22	20
0.38	0.66	1.52	0.33	2.75	0.63	0.72	1.23	0.93	**0.92**	*0.65*	0.15	20
0.82	1.31	1.58	0.63	3.90	0.95	2.01	1.89	1.06	**1.44**	*0.86*	0.19	20
1.00	1.41	3.39	0.89	2.72	0.85	3.35	1.99	0.80	**1.94**	*1.13*	0.25	20
1.20	1.95	0.80	1.25	2.19	1.64	5.60	3.27	1.41	**2.18**	*1.51*	0.34	20
0.68	0.59	1.77	0.50	1.28	0.77	1.17	1.13	0.70	**1.02**	*0.58*	0.13	20
0.96	1.32	1.98	0.88	2.06	1.09	3.37	2.13	0.97	**1.71**	*0.97*	0.22	20
0.99	1.27	1.58	0.47	2.90	1.39	1.35	1.85	2.55	**1.41**	*0.77*	0.17	20
0.87	1.79	1.37	0.99	2.35	1.24	1.62	2.23	4.01	**1.59**	*0.74*	0.16	20
0.61	0.41	0.81	0.44	1.64	0.65	0.83	0.93	1.10	**0.81**	*0.44*	0.10	20
0.82	1.16	1.25	0.63	2.30	1.09	1.27	1.67	2.55	**1.27**	*0.58*	0.13	20
1.02	1.07	1.50	0.56	1.63	1.40	1.46	1.41	0.60	**1.25**	*0.73*	0.16	20
0.74	1.43	1.06	0.99	1.41	1.12	1.99	2.11	1.84	**1.42**	*0.51*	0.12	20
0.68	0.50	0.78	0.36	0.96	0.78	0.90	0.97	0.84	**0.78**	*0.48*	0.11	20
0.81	1.00	1.11	0.63	1.33	1.10	1.45	1.50	1.09	**1.15**	*0.50*	0.11	20
0.93	1.75	1.67	0.57	2.24	1.41	1.72	1.48	0.64	**1.50**	*0.98*	0.22	20
0.76	2.45	1.38	1.16	1.44	0.99	2.07	1.90	2.22	**1.58**	*0.66*	0.15	20
0.85	0.74	0.83	0.40	1.15	0.67	0.87	0.95	0.76	**0.86**	*0.55*	0.12	20
0.84	1.65	1.29	0.71	1.61	1.02	1.55	1.44	1.21	**1.32**	*0.63*	0.14	20
78	100	80	100	97	92	88	100	•	**88.72**	*14.76*	3.48	18
78	•	97	92	92	97	97	100	•	**92.18**	*8.90*	2.16	17
90	•	100	100	97	93	100	100	90	**95.28**	*9.30*	2.19	18
100	100	100	100	100	100	100	100	100	**99.37**	*2.75*	0.63	19
91	100	100	91	100	91	91	91	91	**95.05**	*4.59*	1.03	20
91	100	100	100	100	91	100	100	100	**99.10**	*2.77*	0.62	20
100	96	100	100	100	100	100	100	89	**99.21**	*2.64*	0.60	19
100	100	96	96	100	96	96	100	100	**98.11**	*2.05*	0.47	19
100	100	94	100	100	100	100	100	100	**99.70**	*1.34*	0.30	20

Appendix 6b: Participants in study 2: Classical eyeblink conditioning

	Participant	D1	D2	D3	D4	D5
	Age	19	20	20	22	21
	IQ (short-form WAIS-III)	151	110	133	121	130
	Sex	M	F	M	M	M
	Handed-ness	R	L	L	R	R
WAIS-III Subtest age-scaled scores	Vocabulary	17	11	14	12	11
	Similarities	17	10	14	13	12
	Digit Span	9	9	9	8	8
	Picture Completion	15	9	15	13	15
	Digit-Symbol Coding	8	9	10	10	10
	Block Design	16	16	14	14	18
	ADD	69	39	53	45	55
	NWR time (s)	67	61	88	171	76
	NWR errors	18	9	16	19	13
	WORD spelling (raw score)	35	41	34	36	37
	DAST 1-minute reading	81	89	82	33	74
	DAST 1-minute writing	23	30	29	26	46
	DAST 2-minute spelling	19	31	29	27	28
	Awareness of stimulus relationships (0-3)	2	2	1	1	3
Alpha Responses per CS	Block 1	0.000	0.125	0.125	0.000	0.000
	Block 2	0.000	0.250	0.000	0.000	0.125
	Block 3	0.000	0.000	0.125	0.000	0.000
	Block 4	0.000	0.000	0.000	0.000	0.000
	Block 5	0.250	0.000	0.000	0.000	0.000
	Block 6	0.000	0.000	0.000	0.125	0.000
	CS-alone trials	0.167	0.000	0.167	0.000	0.333
CRs per CS	Block 1	0.125	0.375	0.250	0.000	0.000
	Block 2	0.000	0.875	0.500	0.125	0.125
	Block 3	0.000	0.875	0.750	0.250	0.375
	Block 4	0.375	0.875	0.500	0.000	0.625
	Block 5	0.250	0.875	0.375	0.125	0.500
	Block 6	0.000	1.000	0.625	0.000	0.125
	CS-alone trials	0.333	1.000	0.500	0.333	0.167
	Total CRs produced to all 54 CS presentations	8	45	27	6	15
URs per US	Block 1	0.875	0.375	0.875	1.000	1.000
	Block 2	0.250	0.000	0.875	1.000	0.875
	Block 3	0.500	0.125	1.000	1.000	0.875
	Block 4	0.500	0.125	1.000	0.875	0.500
	Block 5	0.875	0.125	1.000	0.750	0.750
	Block 6	0.625	0.250	0.875	0.625	0.750
	US-alone trials	0.667	1.000	1.000	0.667	1.000
	Block 1	0.250	0.000	0.000	0.000	0.000
	Block 2	0.125	0.125	0.000	0.125	0.125

D6	D7	D8	D9	D10	D11	D12	D13	D14	D15
20	21	22	21	21	21	21	20	21	24
113	126	140	111	115	103	128	130	108	121
F	M	M	M	F	F	M	M	M	F
R	R	R	R	R	L	R	R	R	L
12	11	14	13	12	12	13	13	11	15
13	15	17	13	15	12	15	18	10	15
11	10	15	9	7	7	10	10	9	10
12	13	13	7	12	12	11	13	12	9
8	9	9	6	8	10	10	8	9	12
11	15	16	14	10	6	16	12	12	13
52	98	51	64	42	46	61	20	46	53
65	90	98	71	86	58	52	67	67	78
8	15	10	11	11	17	13	9	10	11
46	35	38	43	39	38	41	40	39	46
103	65	108	77	84	109	102	95	90	87
19	30	29	28	33	34	32	31	34	31
27	28	27	30	32	29	33	32	31	35
3	3	3	1	0	2	2	2	2	3
0.250	0.000	0.125	0.250	0.250	0.375	0.000	0.000	0.125	0.375
0.125	0.000	0.000	0.000	0.250	0.250	0.000	0.000	0.000	0.000
0.125	0.000	0.000	0.125	0.250	0.125	0.000	0.125	0.000	0.250
0.000	0.000	0.000	0.000	0.250	0.000	0.000	0.125	0.000	0.125
0.125	0.000	0.000	0.125	0.000	0.125	0.000	0.000	0.125	0.000
0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.125	0.000
0.000	0.167	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
0.625	0.750	0.000	0.250	0.125	0.500	0.375	0.375	0.125	0.750
0.875	0.875	0.250	0.375	0.375	0.500	0.375	0.625	0.000	0.875
0.750	0.625	0.000	0.750	0.750	0.625	0.750	0.625	0.125	0.750
1.000	0.625	0.000	0.875	0.750	0.750	0.750	0.625	0.500	0.750
0.875	0.625	0.250	0.625	0.500	0.750	0.375	0.375	0.625	0.875
1.000	0.625	0.750	0.875	0.875	0.375	0.375	0.625	0.625	0.875
1.000	0.667	0.667	0.833	0.500	0.833	0.833	0.500	0.500	0.833
47	37	14	35	30	33	29	29	19	44
0.375	0.500	1.000	1.000	0.875	1.000	0.625	0.500	0.875	0.625
0.000	0.250	0.750	0.875	0.875	0.875	0.625	0.375	1.000	0.500
0.000	0.375	0.625	1.000	0.875	1.000	0.500	0.250	0.750	0.750
0.000	0.375	0.500	0.875	0.750	1.000	0.375	0.250	0.375	0.625
0.250	0.500	0.250	1.000	0.875	0.875	0.750	0.000	0.375	0.625
0.000	0.625	0.500	1.000	0.875	0.625	0.875	0.000	0.500	0.375
1.000	1.000	0.667	1.000	0.833	1.000	1.000	1.000	1.000	1.000
0.125	0.000	0.000	0.125	0.125	0.500	0.000	0.125	0.000	0.125
0.250	0.250	0.000	0.000	0.250	0.250	0.000	0.250	0.000	0.000

D16	D17	D18	Mean	*St. Dev.*	St. Err.	n	C1	C2	C3
23	20	18	**20.83**	*1.38*	0.33	18	23	21	24
130 M R	121 M R	121 M R	**122.89**	*12.06*	2.84	18	130 M R	106 M R	111 M R
17	13	11	**12.89**	*1.91*	0.45	18	15	15	14
12	13	12	**13.67**	*2.28*	0.54	18	14	10	8
8	11	10	**9.44**	*1.82*	0.43	18	13	12	12
12	15	13	**12.28**	*2.22*	0.52	18	12	8	12
19	7	5	**9.28**	*2.93*	0.69	18	12	11	11
15	11	16	**13.61**	*2.89*	0.68	18	15	11	13
31	76	73	**54.11**	*17.88*	4.21	18	53	15	47
69	82	127	**81.83**	*28.22*	6.65	18	50	48	74
15	16	16	**13.17**	*3.42*	0.81	18	6	4	7
36	37	34	**38.61**	*3.68*	0.87	18	46	44	45
119	83	53	**85.22**	*20.81*	4.90	18	133	109	98
28	33	29	**30.28**	*5.46*	1.29	18	34	43	39
24	31	25	**28.78**	*3.73*	0.88	18	40	38	35
2	3	2	**2.06**	*0.87*	0.21	18	3	1	2
0.500	0.250	0.250	**0.17**	*0.15*	0.04	18	0.125	0.125	0.000
0.375	0.125	0.125	**0.09**	*0.12*	0.03	18	0.250	0.000	0.000
0.000	0.125	0.125	**0.08**	*0.09*	0.02	18	0.000	0.000	0.000
0.250	0.000	0.000	**0.04**	*0.09*	0.02	18	0.000	0.000	0.125
0.250	0.000	0.000	**0.06**	*0.09*	0.02	18	0.000	0.000	0.000
0.250	0.000	0.000	**0.03**	*0.07*	0.02	18	0.000	0.125	0.000
0.167	0.000	0.000	**0.06**	*0.10*	0.02	18	0.000	0.000	0.000
0.500	0.000	0.000	**0.28**	*0.26*	0.06	18	0.000	0.000	0.000
0.750	0.000	0.125	**0.42**	*0.33*	0.08	18	0.000	0.125	0.375
0.875	0.000	0.125	**0.50**	*0.33*	0.08	18	0.250	0.000	0.250
1.000	0.000	0.000	**0.56**	*0.35*	0.08	18	0.000	0.000	0.375
0.750	0.125	0.375	**0.51**	*0.25*	0.06	18	0.125	0.000	0.125
1.000	0.250	0.125	**0.56**	*0.35*	0.08	18	0.000	0.000	0.125
0.833	0.667	0.500	**0.64**	*0.24*	0.06	18	0.000	0.000	0.333
44	7	9	**26.56**	*14.18*	3.34	18	3	1	12
0.625	1.000	1.000	**0.78**	*0.23*	0.06	18	1.000	1.000	1.000
0.375	1.000	1.000	**0.64**	*0.35*	0.08	18	1.000	0.500	0.625
0.250	1.000	1.000	**0.66**	*0.34*	0.08	18	1.000	0.375	0.750
0.375	1.000	1.000	**0.58**	*0.32*	0.08	18	1.000	0.000	1.000
0.500	1.000	1.000	**0.64**	*0.33*	0.08	18	1.000	0.000	1.000
0.375	1.000	0.750	**0.59**	*0.30*	0.07	18	0.625	0.125	1.000
0.833	1.000	0.833	**0.92**	*0.13*	0.03	18	1.000	0.500	0.833
0.125	0.250	0.125	**0.10**	*0.13*	0.03	18	0.000	0.125	0.000
0.125	0.000	0.125	**0.11**	*0.10*	0.02	18	0.000	0.000	0.125

C4	C5	C6	C7	C8	C9	C10	C11	C12	C13
22	21	23	22	20	19	21	23	20	23
113	111	99	111	108	101	133	137	119	145
M	M	F	M	F	F	M	M	M	M
L	R	R	R	R	R	R	L	L	R
18	14	11	18	17	12	19	18	12	17
11	13	11	13	12	10	14	12	11	15
11	12	11	10	12	9	14	15	8	17
8	9	8	5	7	8	12	13	12	15
14	11	15	9	7	8	15	14	11	16
11	11	10	11	9	11	12	16	16	15
57	15	50	42	38	14	31	27	17	32
44	43	35	50	48	42	39	49	47	36
7	4	8	2	1	4	5	4	6	1
45	47	44	48	44	48	45	43	44	46
124	119	120	122	94	119	135	123	111	125
40	33	35	32	33	33	31	41	34	34
35	37	36	39	31	40	34	31	34	36
2	2	2	3	2	1	3	1	3	2
0.125	0.500	0.000	0.000	0.375	0.000	0.000	0.125	0.250	0.000
0.125	0.000	0.000	0.000	0.500	0.000	0.125	0.000	0.375	0.000
0.000	0.000	0.125	0.000	0.625	0.000	0.000	0.000	0.000	0.000
0.000	0.000	0.000	0.000	0.125	0.000	0.000	0.125	0.375	0.000
0.000	0.000	0.125	0.000	0.000	0.125	0.000	0.000	0.125	0.000
0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
0.000	0.000	0.000	0.167	0.333	0.000	0.000	0.000	0.167	0.167
0.000	0.125	0.000	0.000	0.250	0.250	0.125	0.125	0.125	0.500
0.000	0.000	0.000	0.375	0.750	0.500	0.000	0.000	0.625	0.625
0.250	0.125	0.000	0.375	0.625	0.750	0.000	0.000	0.750	0.625
0.000	0.000	0.125	0.625	0.625	0.500	0.000	0.125	0.750	0.875
0.000	0.000	0.375	0.500	0.625	0.500	0.000	0.125	0.875	0.750
0.125	0.000	0.375	0.250	0.500	0.625	0.000	0.000	0.625	0.750
0.167	0.167	0.500	0.500	0.667	1.000	0.000	0.167	1.000	0.833
4	3	10	20	31	31	1	4	36	38
0.875	1.000	1.000	0.875	0.875	0.875	1.000	0.875	0.750	0.875
1.000	0.625	1.000	1.000	0.875	0.625	0.875	0.750	0.625	0.875
1.000	1.000	1.000	1.000	1.000	1.000	0.500	1.000	0.875	0.750
1.000	0.625	0.750	0.750	0.875	0.875	0.500	1.000	0.625	0.500
1.000	0.125	1.000	1.000	0.750	0.875	0.125	0.875	0.625	0.625
1.000	0.250	0.875	0.625	1.000	0.875	0.375	0.750	0.750	0.750
1.000	0.833	1.000	1.000	0.833	0.833	0.667	1.000	1.000	0.833
0.125	0.375	0.000	0.000	0.125	0.125	0.125	0.000	0.000	0.000
0.125	0.000	0.000	0.125	0.125	0.125	0.000	0.000	0.000	0.000

C14	C15	C16	Mean	*St. Dev.*	St. Err.	n
21	21	20	**21.50**	*1.41*	0.35	16
115	146	143	**120.50**	*16.01*	4.00	16
F	F	M				
R	R	R				
13	17	17	**15.44**	*2.53*	0.63	16
11	14	18	**12.31**	*2.39*	0.60	16
12	13	13	**12.13**	*2.19*	0.55	16
12	18	9	**10.50**	*3.33*	0.83	16
10	12	8	**11.50**	*2.73*	0.68	16
13	14	17	**12.81**	*2.43*	0.61	16
30	50	65	**36.44**	*16.34*	4.09	16
45	70	40	**47.50**	*10.68*	2.67	16
4	3	4	**4.38**	*2.06*	0.52	16
44	46	49	**45.50**	*1.75*	0.44	16
121	87	128	**116.75**	*13.66*	3.41	16
36	33	38	**35.56**	*3.56*	0.89	16
40	40	37	**36.44**	*3.01*	0.75	16
3	3	2	**2.19**	*0.75*	0.19	16
0.125	0.125	0.000	**0.12**	*0.15*	0.04	16
0.375	0.000	0.000	**0.11**	*0.17*	0.04	16
0.125	0.125	0.125	**0.07**	*0.16*	0.04	16
0.125	0.000	0.000	**0.05**	*0.10*	0.03	16
0.125	0.250	0.125	**0.05**	*0.08*	0.02	16
0.250	0.000	0.000	**0.02**	*0.07*	0.02	16
0.333	0.167	0.000	**0.08**	*0.12*	0.03	16
0.625	0.375	0.000	**0.16**	*0.20*	0.05	16
0.625	1.000	0.000	**0.31**	*0.34*	0.08	16
0.750	1.000	0.000	**0.36**	*0.34*	0.09	16
0.750	1.000	0.000	**0.36**	*0.37*	0.09	16
0.500	1.000	0.000	**0.34**	*0.34*	0.09	16
0.875	0.750	0.000	**0.31**	*0.33*	0.08	16
0.667	0.833	0.167	**0.44**	*0.36*	0.09	16
37	46	1	**17.38**	*16.36*	4.09	16
0.750	0.750	1.000	**0.91**	*0.10*	0.02	16
0.375	0.750	0.625	**0.76**	*0.20*	0.05	16
0.250	0.875	0.500	**0.80**	*0.26*	0.06	16
0.125	0.875	0.625	**0.70**	*0.30*	0.08	16
0.125	0.750	0.250	**0.63**	*0.38*	0.09	16
0.375	0.625	0.250	**0.64**	*0.29*	0.07	16
0.833	1.000	0.833	**0.88**	*0.14*	0.04	16
0.500	0.125	0.000	**0.10**	*0.15*	0.04	16
0.375	0.125	0.125	**0.08**	*0.10*	0.03	16

No Stimulus Responses per trial	Block 3	0.000	0.125	0.000	0.250	0.000
	Block 4	0.000	0.000	0.000	0.000	0.000
	Block 5	0.250	0.000	0.000	0.000	0.000
	Block 6	0.125	0.000	0.000	0.125	0.000
	CS-alone trials	0.167	0.167	0.167	0.167	0.000
	US-alone trials	0.500	0.000	0.167	0.167	0.000
Alpha response onset times (ms post CS onset)	Block 1	•	73	7	•	•
	Block 2	•	118	•	•	133
	Block 3	•	•	8	•	•
	Block 4	•	•	•	•	•
	Block 5	93	•	•	•	•
	Block 6	•	•	•	130	•
	CS-alone trials	80	•	122	•	83
Alpha response peak times (ms post CS onset)	Block 1	•	140	98	•	•
	Block 2	•	158	•	•	194
	Block 3	•	•	108	•	•
	Block 4	•	•	•	•	•
	Block 5	152	•	•	•	•
	Block 6	•	•	•	158	•
	CS-alone trials	116	•	163	•	148
Alpha response peak amplitudes	Block 1	•	44	64	•	•
	Block 2	•	35	•	•	14
	Block 3	•	•	24	•	•
	Block 4	•	•	•	•	•
	Block 5	26	•	•	•	•
	Block 6	•	•	•	11	•
	CS-alone trials	41	•	21	•	21
CR onset times (ms post CS onset)	Block 1	•	255	275	•	•
	Block 2	•	220	289	•	381
	Block 3	•	274	303	•	279
	Block 4	•	295	298	•	294
	Block 5	•	256	342	•	342
	Block 6	•	281	306	•	243
	CS-alone trials	•	307	335	•	370
CR peak times (ms post CS onset)	Block 1	•	317	369	•	•
	Block 2	•	272	417	•	462
	Block 3	•	330	401	•	338
	Block 4	•	358	376	•	366
	Block 5	•	320	446	•	418
	Block 6	•	348	385	•	349
	CS-alone trials	•	374	432	•	437
CR tuning	Deviation of CR peak from 450ms post CS onset in block 2	•	178	34	•	12
	Deviation of CR peak from 450ms post CS onset in block 6	•	102	65	•	101

0.125	0.000	0.000	0.000	0.250	0.000	0.000	0.250	0.125	0.000
0.000	0.000	0.000	0.125	0.125	0.000	0.000	0.125	0.000	0.125
0.000	0.000	0.000	0.000	0.125	0.125	0.000	0.000	0.000	0.000
0.000	0.000	0.000	0.125	0.000	0.000	0.000	0.125	0.000	0.000
0.000	0.000	0.000	0.000	0.167	0.167	0.000	0.000	0.000	0.000
0.000	0.333	0.167	0.000	0.000	0.000	0.000	0.333	0.333	0.167
89	•	147	85	73	99	•	•	92	92
80	•	•	•	29	65	•	•	•	•
96	•	•	85	105	131	•	67	•	84
•	•	•	•	79	•	•	146	•	24
141	•	•	8	•	145	•	•	103	•
•	•	•	•	•	•	•	•	112	•
•	138	•	•	•	•	•	•	•	•
172	•	174	148	194	170	•	•	159	139
175	•	•	•	125	180	•	•	•	•
149	•	•	136	169	246	•	142	•	254
•	•	•	•	166	•	•	197	•	140
197	•	•	81	•	280	•	•	147	•
•	•	•	•	•	•	•	•	173	•
•	236	•	•	•	•	•	•	•	•
34	•	18	43	56	32	•	•	44	34
61	•	•	•	43	32	•	•	•	•
56	•	•	14	55	27	•	17	•	65
•	•	•	•	49	•	•	25	•	55
50	•	•	53	•	15	•	•	26	•
•	•	•	•	•	•	•	•	12	•
•	19	•	•	•	•	•	•	•	•
280	230	•	354	330	228	273	281	176	240
240	258	362	308	295	271	301	255	•	232
256	263	•	293	260	257	337	240	315	235
241	322	•	329	314	259	305	262	319	243
236	340	350	304	306	340	354	258	276	274
269	304	334	304	315	341	244	240	343	229
286	289	429	465	288	327	473	224	403	210
347	308	•	431	401	312	385	350	266	313
298	335	416	370	419	384	409	325	•	301
307	346	•	359	377	395	416	299	398	324
288	406	•	391	420	382	363	322	399	331
283	413	412	362	381	462	435	319	363	391
316	376	420	354	398	437	305	295	435	317
339	357	539	521	384	426	533	298	495	296
152	115	35	80	31	66	41	125	•	149
134	74	30	96	52	13	145	155	•	133

0.125	0.000	0.000	**0.07**	*0.10*	0.02	18	0.250	0.000	0.000
0.375	0.125	0.000	**0.06**	*0.10*	0.02	18	0.000	0.000	0.000
0.000	0.000	0.125	**0.03**	*0.07*	0.02	18	0.000	0.000	0.000
0.375	0.000	0.125	**0.06**	*0.10*	0.02	18	0.000	0.000	0.000
0.333	0.167	0.000	**0.08**	*0.10*	0.02	18	0.167	0.000	0.000
0.333	0.000	0.167	**0.15**	*0.16*	0.04	18	0.000	0.167	0.167
68	36	34	**74.47**	*36.36*	10.50	12	142	49	•
68	69	110	**83.98**	*34.14*	12.07	8	143	•	•
•	83	105	**84.83**	*34.09*	11.36	9	•	•	•
89	•	•	**84.38**	*50.00*	25.00	4	•	•	12
140	•	•	**104.92**	*52.24*	21.33	6	•	•	•
113	•	•	**118.17**	*10.25*	5.92	3	•	44	•
147	•	•	**114.00**	*31.01*	13.87	5	•	•	•
149	145	85	**147.74**	*30.95*	8.93	12	312	119	•
126	122	162	**155.02**	*27.92*	9.87	8	254	•	•
•	111	151	**162.83**	*52.86*	17.62	9	•	•	•
155	•	•	**164.25**	*24.20*	12.10	4	•	•	73
207	•	•	**177.25**	*67.26*	27.46	6	•	•	•
169	•	•	**166.50**	*7.70*	4.44	3	•	90	•
200	•	•	**172.60**	*46.56*	20.82	5	•	•	•
50	17	22	**37.93**	*14.88*	4.30	12	15	27	•
46	13	15	**32.35**	*17.52*	6.20	8	18	•	•
•	11	13	**31.28**	*21.23*	7.08	9	•	•	•
47	•	•	**44.00**	*13.11*	6.56	4	•	•	12
58	•	•	**37.85**	*17.76*	7.25	6	•	•	•
41	•	•	**21.17**	*16.75*	9.67	3	•	20	•
30	•	•	**26.30**	*9.28*	4.15	5	•	•	•
287	•	•	**267.37**	*47.08*	13.59	12	•	•	•
270	•	•	**283.21**	*47.49*	13.17	13	•	•	293
257	•	•	**274.44**	*29.90*	8.29	13	•	•	245
235	•	•	**285.84**	*33.49*	9.29	13	•	•	307
223	•	•	**300.14**	*45.72*	12.22	14	•	•	210
243	•	•	**285.41**	*40.77*	10.90	14	•	•	318
254	•	•	**332.84**	*84.54*	22.60	14	•	•	309
389	•	•	**349.06**	*47.56*	13.73	12	•	•	•
363	•	•	**366.84**	*57.47*	15.94	13	•	•	365
337	•	•	**355.85**	*38.12*	10.57	13	•	•	311
307	•	•	**362.19**	*39.93*	11.07	13	•	•	339
290	•	•	**378.27**	*57.60*	15.39	14	•	•	276
308	•	•	**360.33**	*49.15*	13.14	14	•	•	373
338	•	•	**412.06**	*84.68*	22.63	14	•	•	364
88	•	•	**85.01**	*54.47*	15.11	13	•	•	85
142	•	•	**95.44**	*45.95*	12.75	13	•	•	77

0.000	0.125	0.375	0.000	0.000	0.375	0.000	0.125	0.250	0.000
0.000	0.000	0.125	0.000	0.125	0.125	0.125	0.125	0.125	0.000
0.000	0.000	0.250	0.000	0.000	0.000	0.000	0.000	0.125	0.000
0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.125	0.000
0.000	0.000	0.000	0.167	0.167	0.167	0.167	0.000	0.167	0.000
0.000	0.167	0.167	0.000	0.167	0.167	0.000	0.000	0.333	0.167
57	85	•	•	68	•	•	4	68	•
57	•	•	•	66	•	39	•	66	•
•	•	61	•	72	•	•	•	•	•
•	•	•	•	79	•	•	75	66	•
•	•	38	•	•	43	•	•	97	•
•	•	•	•	•	•	•	•	•	•
•	•	•	72	89	•	•	•	81	81
119	130	•	•	110	•	•	90	116	•
97	•	•	•	112	•	88	•	115	•
•	•	127	•	120	•	•	•	•	•
•	•	•	•	125	•	•	162	153	•
•	•	122	•	•	111	•	•	147	•
•	•	•	•	•	•	•	•	•	•
•	•	•	138	116	•	•	•	115	129
22	18	•	•	27	•	•	49	51	•
11	•	•	•	21	•	12	•	14	•
•	•	27	•	20	•	•	•	•	•
•	•	•	•	14	•	•	38	65	•
•	•	16	•	•	26	•	•	66	•
•	•	•	•	•	•	•	•	•	•
•	•	•	15	13	•	•	•	11	22
•	•	•	•	231	173	•	•	•	315
•	•	•	294	241	302	•	•	266	304
•	•	•	239	288	334	•	•	236	333
•	•	•	308	271	307	•	•	290	331
•	•	•	340	320	290	•	•	269	326
•	•	•	394	315	331	•	•	283	302
•	•	•	416	364	375	•	•	304	290
•	•	•	•	306	272	•	•	•	392
•	•	•	344	361	382	•	•	352	385
•	•	•	344	356	412	•	•	313	418
•	•	•	444	359	388	•	•	370	454
•	•	•	428	405	385	•	•	339	462
•	•	•	470	433	412	•	•	351	420
•	•	•	513	459	451	•	•	375	401
•	•	•	106	89	68	•	•	98	65
•	•	•	20	17	38	•	•	99	31

0.500	0.125	0.250	**0.15**	*0.17*	0.04	16
0.375	0.000	0.000	**0.07**	*0.10*	0.03	16
0.375	0.125	0.125	**0.06**	*0.11*	0.03	16
0.375	0.000	0.000	**0.03**	*0.10*	0.02	16
0.333	0.000	0.000	**0.08**	*0.11*	0.03	16
0.667	0.167	0.167	**0.16**	*0.17*	0.04	16
94	71	•	**70.86**	*36.99*	12.33	9
73	•	•	**73.94**	*35.79*	14.61	6
39	118	53	**68.64**	*30.13*	13.47	5
45	•	•	**55.47**	*27.62*	12.35	5
139	44	91	**75.33**	*40.47*	16.52	6
77	•	•	**60.25**	*22.98*	16.25	2
141	57	•	**86.75**	*28.69*	11.71	6
153	146	•	**143.81**	*65.79*	21.93	9
143	•	•	**134.99**	*61.30*	25.03	6
107	166	115	**126.92**	*23.02*	10.29	5
92	•	•	**121.00**	*38.29*	17.13	5
193	102	164	**139.83**	*34.76*	14.19	6
132	•	•	**111.00**	*29.70*	21.00	2
209	112	•	**136.42**	*36.93*	15.08	6
14	12	•	**26.06**	*14.46*	4.82	9
51	•	•	**21.14**	*15.25*	6.22	6
72	26	13	**31.68**	*23.22*	10.38	5
11	•	•	**28.07**	*23.64*	10.57	5
50	24	23	**34.08**	*19.49*	7.96	6
36	•	•	**27.75**	*10.96*	7.75	2
59	20	•	**23.17**	*17.82*	7.28	6
253	359	•	**265.98**	*72.63*	32.48	5
269	305	•	**284.35**	*23.08*	8.16	8
245	285	•	**275.65**	*41.02*	14.50	8
291	275	•	**297.39**	*19.86*	7.02	8
268	322	•	**293.07**	*43.02*	15.21	8
302	299	•	**318.05**	*33.88*	11.98	8
313	310	•	**335.10**	*44.39*	15.69	8
308	422	•	**339.93**	*63.73*	28.50	5
324	350	•	**357.83**	*20.08*	7.10	8
313	337	•	**350.47**	*42.97*	15.19	8
365	344	•	**382.83**	*43.77*	15.48	8
355	362	•	**376.37**	*57.28*	20.25	8
363	361	•	**397.79**	*42.09*	14.88	8
397	356	•	**414.50**	*55.07*	19.47	8
126	100	•	**92.17**	*20.08*	7.10	8
87	89	•	**57.08**	*34.09*	12.05	8

	Decrease in deviation of CR peak from 450ms post CS onset from block 2 to block 6	•	77	-32	•	-89
CR peak amplitudes	Block 1	•	45	50	•	•
	Block 2	•	45	51	108	47
	Block 3	•	38	40	21	38
	Block 4	57	36	49	•	35
	Block 5	39	30	29	16	25
	Block 6	•	39	34	•	23
	CS-alone trials	45	38	46	71	22
UR onset times (ms post US onset)	Block 1	80	56	63	52	68
	Block 2	70	•	102	74	58
	Block 3	75	62	110	67	49
	Block 4	109	90	134	53	99
	Block 5	48	83	101	69	68
	Block 6	75	129	122	81	75
	US-alone trials	133	67	86	84	70
UR peak times (ms post US onset)	Block 1	133	133	111	115	118
	Block 2	107	•	161	125	108
	Block 3	110	105	163	111	104
	Block 4	149	149	185	109	156
	Block 5	86	147	150	126	132
	Block 6	116	216	181	157	129
	US-alone trials	170	135	139	165	123
UR peak amplitudes	Block 1	92	58	101	144	90
	Block 2	82	•	59	87	64
	Block 3	50	59	50	89	42
	Block 4	51	41	46	53	29
	Block 5	56	46	31	46	25
	Block 6	46	19	23	29	20
	US-alone trials	57	55	59	88	53
No Stimulus Resonse onset times (ms post trial start)	Block 1	360	•	•	•	•
	Block 2	304	231	•	293	486
	Block 3	•	366	•	408	•
	Block 4	•	•	•	•	•
	Block 5	178	•	•	•	•
	Block 6	485	•	•	413	•
	CS-alone trials	339	425	462	147	•
	US-alone trials	461	•	410	762	•
No Stimulus Response peak times (ms post trial start)	Block 1	415	•	•	•	•
	Block 2	362	337	•	363	534
	Block 3	•	446	•	481	•
	Block 4	•	•	•	•	•
	Block 5	209	•	•	•	•
	Block 6	531	•	•	443	•
	CS-alone trials	370	503	535	188	•
	US-alone trials	490	•	465	834	•
	Block 1	68	•	•	•	•
	Block 2	75	31	•	20	51

18	41	5	-16	-21	53	-103	-30	•	17
73	60	•	34	56	33	51	36	29	58
62	58	17	58	66	25	49	22	•	59
61	47	•	43	49	26	42	24	45	65
56	32	•	53	48	21	40	16	45	66
52	27	11	35	35	20	34	14	56	73
47	20	16	45	35	12	31	16	42	52
57	42	18	41	35	21	41	18	59	66
68	55	97	63	70	59	64	60	61	79
•	80	54	65	67	72	48	42	60	65
•	43	109	50	72	111	128	56	49	61
•	62	117	86	78	143	87	86	65	68
125	76	154	86	68	155	89	•	69	66
•	76	145	86	100	37	62	•	72	66
68	76	140	68	65	76	90	70	72	54
104	119	176	109	126	106	141	131	142	135
•	147	122	113	126	126	117	117	117	128
•	115	177	109	122	173	179	111	128	137
•	119	230	128	136	188	159	169	126	126
176	137	257	128	151	193	139	•	144	105
•	120	226	141	166	108	106	•	141	123
106	125	188	124	140	141	137	159	125	147
99	68	48	58	95	67	83	59	107	44
•	41	20	72	90	43	58	43	84	59
•	53	12	69	81	24	44	31	65	60
•	42	16	57	74	20	34	31	50	65
27	35	15	54	75	14	30	•	60	68
•	26	14	46	79	15	32	•	63	73
74	60	17	75	98	53	47	32	71	95
426	•	•	297	123	341	•	210	•	106
316	195	•	•	234	234	•	219	•	•
240	•	•	•	254	•	•	276	344	•
•	•	•	496	466	•	•	408	•	461
•	•	•	•	134	449	•	•	•	•
•	•	•	352	•	•	•	366	•	•
•	•	•	•	338	462	•	•	•	•
•	437	875	•	•	•	•	245	201	337
491	•	•	416	228	418	•	265	•	197
381	256	•	•	303	307	•	263	•	•
298	•	•	•	336	•	•	330	395	•
•	•	•	582	521	•	•	444	•	530
•	•	•	•	221	524	•	•	•	•
•	•	•	419	•	•	•	412	•	•
•	•	•	•	415	535	•	•	•	•
•	513	934	•	•	•	•	291	256	423
69	•	•	25	60	29	•	24	•	74
60	38	•	•	42	27	•	25	•	•

-54	•	•	-10.44	*52.95*	14.69	13	•	•	8
57	•	•	48.51	*13.34*	3.85	12	•	•	•
61	•	40	51.17	*21.89*	5.65	15	•	21	28
62	•	35	42.26	*13.35*	3.45	15	13	•	13
58	•	•	43.64	*14.51*	3.88	14	•	•	16
54	16	13	32.09	*17.31*	4.08	18	11	•	22
54	17	19	31.36	*14.11*	3.53	16	•	•	28
49	24	14	39.21	*17.24*	4.06	18	•	•	22
60	64	62	65.55	*10.74*	2.53	18	68	63	58
58	58	46	63.66	*14.47*	3.62	16	67	63	89
98	58	54	73.48	*26.78*	6.50	17	79	74	64
80	60	73	87.59	*25.45*	6.17	17	78	•	70
44	61	83	85.03	*32.35*	7.85	17	75	•	57
73	64	72	83.38	*27.72*	6.93	16	81	168	71
60	65	83	79.13	*22.73*	5.36	18	69	85	88
119	125	125	126.01	*16.93*	3.99	18	115	104	96
118	127	98	122.20	*15.04*	3.76	16	125	98	121
148	122	109	130.68	*26.81*	6.50	17	124	104	98
117	122	113	145.87	*32.61*	7.91	17	132	•	107
111	120	119	142.39	*38.81*	9.41	17	135	•	87
114	122	110	142.12	*37.48*	9.37	16	130	212	109
111	124	124	137.88	*21.25*	5.01	18	141	135	129
89	74	81	80.87	*24.50*	5.77	18	30	59	18
55	37	52	59.02	*20.03*	5.01	16	31	46	36
53	35	57	51.42	*19.51*	4.73	17	33	17	17
24	48	48	42.82	*15.79*	3.83	17	22	•	15
56	64	45	43.94	*18.43*	4.47	17	21	•	22
41	45	29	37.42	*20.18*	5.04	16	12	19	26
94	75	51	64.04	*21.87*	5.16	18	26	30	24
341	370	236	281.03	*108.18*	34.21	10	•	313	•
296	•	304	282.73	*79.32*	23.92	11	•	•	276
463	•	•	335.71	*83.63*	31.61	7	448	•	•
291	465	•	431.17	*74.34*	30.35	6	•	•	•
•	•	417	294.38	*161.58*	80.79	4	•	•	•
344	•	351	385.17	*54.90*	22.41	6	•	•	•
385	380	•	367.19	*101.36*	35.84	8	271	•	•
620	•	804	515.18	*237.63*	75.15	10	•	681	541
404	464	294	359.08	*103.61*	32.76	10	•	386	•
359	•	366	348.18	*74.94*	22.59	11	•	•	346
559	•	•	406.21	*94.13*	35.58	7	556	•	•
363	532	•	495.39	*78.45*	32.03	6	•	•	•
•	•	469	355.63	*164.29*	82.15	4	•	•	•
409	•	396	435.00	*49.52*	20.22	6	•	•	•
445	474	•	433.13	*114.55*	40.50	8	413	•	•
697	•	868	577.08	*241.60*	76.40	10	•	732	599
60	39	13	46.03	*22.41*	7.09	10	•	57	•
61	•	26	41.36	*17.97*	5.42	11	•	•	26

•	•	•	87	72	30	•	•	-1	35
•	74	•	•	33	23	25	59	•	36
•	•	•	25	49	35	•	•	95	49
25	18	•	34	36	26	•	•	75	39
•	•	45	31	37	23	•	11	78	50
•	•	49	16	49	33	•	33	65	46
21	•	33	18	33	24	•	•	74	42
16	12	26	36	49	27	•	26	84	36
79	75	42	52	69	62	81	63	48	46
74	49	41	56	74	56	70	65	74	69
90	61	54	61	51	93	48	62	66	29
88	57	71	56	77	58	71	70	92	49
78	157	83	67	90	73	80	95	64	44
82	84	79	61	110	86	70	63	120	53
92	75	82	58	54	76	89	66	57	58
119	132	92	113	123	118	132	101	104	103
113	108	93	110	140	135	109	101	147	147
131	110	95	111	118	152	102	100	116	109
131	105	109	106	130	110	108	106	137	105
105	286	126	120	146	126	114	139	113	112
125	132	120	106	156	140	116	116	178	116
131	128	127	126	106	131	134	105	121	110
79	67	96	37	56	89	52	77	95	93
55	36	59	98	50	58	29	83	78	72
46	22	58	48	50	40	27	69	60	66
39	19	47	39	56	38	20	50	70	56
40	50	37	34	52	40	11	36	59	51
34	22	26	23	37	33	12	25	76	55
50	81	46	73	77	68	32	66	97	92
254	280	•	•	380	491	315	•	•	•
351	•	•	351	444	448	•	•	•	•
•	465	293	•	•	141	•	228	467	•
•	•	121	•	477	477	325	332	269	•
•	•	387	•	•	•	•	•	182	•
•	•	•	•	•	•	•	•	103	•
•	•	•	417	404	459	143	•	166	•
•	751	812	•	107	233	•	•	575	864
294	343	•	•	494	570	366	•	•	•
437	•	•	394	508	522	•	•	•	•
•	590	367	•	•	231	•	277	559	•
•	•	204	•	633	551	376	363	357	•
•	•	461	•	•	•	•	•	265	•
•	•	•	•	•	•	•	•	193	•
•	•	•	476	559	546	203	•	257	•
•	828	883	•	216	298	•	•	679	1019
17	61	•	•	45	32	24	•	•	•
28	•	•	26	19	35	•	•	•	•

39	11	•	**35.09**	*30.90*	10.93	8
73	52	•	**46.73**	*20.71*	7.32	8
73	49	•	**47.17**	*24.13*	8.04	9
78	48	•	**36.83**	*22.54*	6.80	11
79	38	•	**40.75**	*23.30*	7.37	10
78	39	•	**40.11**	*20.31*	6.13	11
72	31	•	**37.45**	*19.89*	6.29	10
80	42	31	**37.36**	*22.16*	6.15	13
60	64	64	**62.09**	*11.26*	2.81	16
55	120	61	**67.66**	*18.00*	4.50	16
80	161	63	**70.89**	*28.81*	7.20	16
20	155	63	**71.67**	*28.76*	7.43	15
80	169	53	**84.25**	*34.79*	8.98	15
76	95	61	**84.95**	*28.27*	7.07	16
107	64	67	**74.12**	*15.22*	3.80	16
113	95	135	**112.14**	*13.64*	3.41	16
108	175	107	**121.01**	*22.17*	5.54	16
108	212	94	**117.59**	*29.27*	7.32	16
81	209	109	**118.88**	*28.87*	7.46	15
130	217	103	**137.23**	*50.49*	13.04	15
135	137	140	**135.42**	*27.33*	6.83	16
158	98	145	**126.58**	*15.89*	3.97	16
86	82	73	**67.97**	*24.03*	6.01	16
71	36	41	**54.84**	*20.67*	5.17	16
83	34	36	**44.08**	*19.34*	4.83	16
98	25	20	**40.97**	*22.90*	5.91	15
94	31	16	**39.47**	*20.57*	5.31	15
82	48	13	**33.96**	*21.40*	5.35	16
95	72	59	**61.70**	*24.77*	6.19	16
312	414	•	**344.88**	*78.21*	27.65	8
250	318	128	**320.71**	*105.17*	37.18	8
291	224	270	**314.04**	*118.47*	39.49	9
284	•	•	**326.43**	*124.28*	46.97	7
293	400	258	**303.93**	*91.15*	40.76	5
386	•	•	**244.67**	*200.35*	141.67	2
243	•	•	**300.43**	*126.81*	47.93	7
568	618	888	**603.43**	*246.99*	74.47	11
382	492	•	**415.88**	*92.85*	32.83	8
322	400	228	**394.58**	*97.36*	34.42	8
355	293	369	**399.48**	*134.69*	44.90	9
349	•	•	**404.71**	*142.45*	53.84	7
364	449	319	**371.67**	*83.91*	37.53	5
462	•	•	**327.50**	*190.21*	134.50	2
304	•	•	**393.93**	*141.92*	53.64	7
636	681	953	**684.02**	*249.96*	75.36	11
62	46	•	**42.98**	*17.09*	6.04	8
53	47	39	**34.08**	*11.54*	4.08	8

No Stimulus Response Peak Amplitues	Block 3	•	15	•	72	•
	Block 4	•	•	•	•	•
	Block 5	50	•	•	•	•
	Block 6	43	•	•	32	•
	CS-alone trials	65	35	55	54	•
	US-alone trials	52	•	37	111	•
Extinction Session	Alpha Responses per Trial	0.1	0.2	0.0	0.0	0.0
	CRs per Trial	0.3	0.3	0.3	0.0	0.2
	No Stimulus Responses per Trial	0.1	0.0	0.0	0.1	0.0
	Alpha Response onset times (ms post CS-onset)	47	66	•	•	•
	Alpha Response peak times (ms post CS-onset)	74	121	•	•	•
	Alpha Response peak amplitudes	39	19	•	•	•
	CR onset times (ms post CS-onset)	382	392	391	•	277
	CR peak times (ms post CS-onset)	•	470	484	•	370
	CR peak amplitudes	34	38	25	•	15
	No Stimulus Response onset times (ms post trial start)	162	•	•	237	•
	No Stimulus Response peak times (ms post trial start)	195	•	•	299	•
	No Stimulus Response peak amplitudes	40	•	•	14	•
CS-alone baseline session trials	Alpha Responses per Trial	0.0	0.0	0.2	0.0	0.0
	CRs per Trial	0.2	0.2	0.0	0.0	0.0
	No Stimulus Responses per Trial	0.2	0.2	0.0	0.0	0.0
	Total responses per trial	2	2	1	0	0
	Alpha Response onset times (ms post CS-onset)	•	•	36	•	•
	Alpha Response peak times (ms post CS-onset)	•	•	106	•	•
	Alpha Response peak amplitudes	•	•	14	•	•
	CR onset times (ms post CS-onset)	386	508	•	•	•
	CR peak times (ms post CS-onset)	530	561	•	•	•

11	•	•	•	32	•	•	18	57	•
•	•	•	55	52	•	•	11	•	26
•	•	•	•	39	12	•	•	•	•
•	•	•	55	•	•	•	13	•	•
•	•	•	•	54	32	•	•	•	•
•	37	11	•	•	•	•	18	41	45
0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0
0.3	0.7	0.1	0.5	0.7	0.1	0.2	0.3	0.5	0.6
0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.2
•	•	•	•	•	34	•	•	28	•
•	•	•	•	•	125	•	•	129	•
•	•	•	•	•	16	•	•	16	•
323	371	472	308	362	317	410	276	321	339
372	448	552	363	446	385	468	342	407	437
37	22	16	30	37	14	29	15	37	46
•	207	•	•	164	•	•	423	252	218
•	250	•	•	251	•	•	472	334	284
•	20	•	•	42	•	•	11	58	39
0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2
0.2	0.0	0.0	0.2	0.2	0.0	0.0	0.2	0.8	0.2
0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0
1	0	0	1	1	0	0	3	5	2
•	•	•	•	•	•	•	•	15	83
•	•	•	•	•	•	•	•	55	137
•	•	•	•	•	•	•	•	24	12
337	•	•	223	418	•	•	164	364	213
388	•	•	289	505	•	•	247	414	303

59	•	•	**37.64**	*24.74*	9.35	7	13	•	•
58	19	•	**36.89**	*20.62*	8.42	6	•	•	•
•	•	17	**29.38**	*17.82*	8.91	4	•	•	•
56	•	21	**36.72**	*17.83*	7.28	6	•	•	•
57	52	•	**50.50**	*11.22*	3.97	8	41	•	•
59	•	47	**45.72**	*27.14*	8.58	10	•	12	11
0.0	0.0	0.0	**0.03**	*0.06*	0.01	18	0.0	0.1	0.0
0.5	0.0	0.0	**0.31**	*0.23*	0.05	18	0.1	0.0	0.0
0.2	0.0	0.2	**0.07**	*0.08*	0.02	18	0.0	0.0	0.1
•	•	•	**43.63**	*16.60*	8.30	4	•	140	•
•	•	•	**112.13**	*25.65*	12.83	4	•	198	•
•	•	•	**22.38**	*11.15*	5.57	4	•	18	•
398	•	•	**355.89**	*53.99*	13.94	15	389	•	•
457	•	•	**428.70**	*58.18*	15.55	14	•	•	•
49	•	•	**29.58**	*11.46*	2.96	15	10	•	•
328	•	234	**247.17**	*82.46*	27.49	9	•	•	371
385	•	291	**306.72**	*81.96*	27.32	9	•	•	429
54	•	36	**34.78**	*16.58*	5.53	9	•	•	16
0.2	0.0	0.0	**0.04**	*0.09*	0.02	18	0.0	0.0	0.0
0.2	0.0	0.0	**0.13**	*0.19*	0.05	18	0.0	0.0	0.0
0.0	0.0	0.0	**0.04**	*0.11*	0.03	18	0.0	0.2	0.2
2	0	0	**1.11**	*1.37*	0.32	18	0	1	1
70	•	•	**51.00**	*31.12*	15.56	4	•	•	•
161	•	•	**114.75**	*45.76*	22.88	4	•	•	•
58	•	•	**27.00**	*21.32*	10.66	4	•	•	•
268	•	•	**320.11**	*111.48*	37.16	9	•	•	•
331	•	•	**396.47**	*114.01*	38.00	9	•	•	•

•	63	21	•	•	33	•	52	79	•
•	•	19	•	51	33	22	45	84	•
•	•	12	•	•	•	•	•	39	•
•	•	•	•	•	•	•	•	62	•
•	•	•	55	41	28	30	•	80	•
•	51	36	•	18	17	•	•	90	69
0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.9	0.3
0.0	0.1	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0
•	•	•	•	•	•	•	88	•	•
•	•	•	•	•	•	•	115	•	•
•	•	•	•	•	•	•	15	•	•
•	•	•	•	•	462	•	•	312	288
•	•	•	•	•	533	•	•	380	415
•	•	•	•	•	20	•	•	72	26
•	250	176	•	•	497	•	•	•	•
•	292	247	•	•	559	•	•	•	•
•	11	18	•	•	15	•	•	•	•
0.2	0.0	0.0	0.0	0.2	0.0	0.2	0.0	0.2	0.0
0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2
0.0	0.0	0.0	0.0	0.2	0.2	0.0	0.0	0.0	0.0
1	0	0	0	2	1	1	0	1	1
110	•	•	•	147	•	94	•	54	•
138	•	•	•	218	•	151	•	117	•
21	•	•	•	37	•	30	•	17	•
•	•	•	•	•	•	•	•	•	576
•	•	•	•	•	•	•	•	•	626

49	31	39	**42.15**	*20.60*	6.87	9
51	•	•	**43.52**	*22.07*	8.34	7
53	15	11	**25.93**	*18.86*	8.43	5
45	•	•	**53.50**	*12.02*	8.50	2
40	•	•	**45.00**	*17.78*	6.72	7
58	25	14	**36.43**	*26.76*	8.07	11
0.1	0.1	0.1	**0.03**	*0.05*	0.01	16
0.8	1.0	0.1	**0.23**	*0.36*	0.09	16
0.5	0.1	0.0	**0.06**	*0.13*	0.03	16
74	17	31	**70.00**	*48.91*	21.87	5
130	65	95	**120.60**	*49.64*	22.20	5
39	23	13	**21.60**	*10.43*	4.66	5
336	311	467	**366.41**	*73.98*	27.96	7
389	378	•	**418.83**	*65.67*	29.37	5
47	25	18	**31.03**	*21.24*	8.03	7
272	224	•	**298.40**	*116.83*	47.70	6
321	256	•	**350.67**	*121.30*	49.52	6
45	39	•	**24.07**	*14.37*	5.87	6
0.2	0.2	0.0	**0.08**	*0.10*	0.03	16
0.6	0.2	0.0	**0.06**	*0.16*	0.04	16
0.4	0.2	0.0	**0.09**	*0.13*	0.03	16
6	3	0	**1.13**	*1.54*	0.39	16
67	14	•	**81.00**	*46.43*	18.96	6
146	92	•	**143.67**	*42.42*	17.32	6
30	46	•	**30.17**	*10.53*	4.30	6
293	156	•	**341.67**	*214.19*	123.66	3
367	207	•	**400.00**	*211.44*	122.08	3

	CR peak amplitudes	49	19	•	•	•
Calibration session	Maximum amplitude recorded in calibration	3413	5952	2178	4727	1636
	URs per trial	0.8	0.8	1.0	1.0	0.8
	No Stimulus Responses per trial	0.2	0.2	0.0	0.4	0.0
	Total responses (in the 5 session trial)	5	5	5	7	4
	UR onset times (ms post US onset)	103	60	66	84	64
	UR peak times (ms post US onset)	162	105	117	135	111
	UR peak amplitudes	85	94	83	95	97
	No Stimulus Response onset times (ms post trial start)	404	279	•	595	•
	No Stimulus Response peak times (ms post trial start)	443	343	•	651	•
	No Stimulus Response peak amplitudes	63	44	•	26	•

27	•	•	69	52	•	•	12	25	46
1377	2627	3047	2090	7715	4629	2178	2114	3042	1719
0.6	1.0	1.0	0.8	1.0	0.8	1.0	1.0	0.8	0.6
0.2	0.2	0.4	0.6	0.0	0.4	0.0	0.4	0.4	0.0
4	6	7	7	5	6	5	7	6	3
65	65	93	66	64	64	65	69	60	42
100	110	177	107	127	104	124	124	100	106
94	93	86	88	98	96	88	92	85	94
171	295	402	461	•	507	•	245	598	•
243	359	462	511	•	578	•	291	651	•
61	59	22	69	•	24	•	39	27	•

22	•	•	**35.64**	*18.98*	6.33	9	•	•	•
2388	1494	2104	**3023.89**	*1705.19*	401.92	18	830	1348	1294
0.8	1.0	0.6	**0.86**	*0.15*	0.04	18	1.0	1.0	1.0
0.2	0.2	0.2	**0.22**	*0.18*	0.04	18	0.0	0.0	0.0
5	6	4	**5.39**	*1.20*	0.28	18	5	5	5
57	63	71	**67.81**	*13.59*	3.20	18	75	63	61
100	122	126	**119.63**	*21.12*	4.98	18	136	104	97
93	87	76	**90.12**	*5.82*	1.37	18	77	90	66
442	799	401	**430.55**	*169.03*	46.88	13	•	•	•
462	865	458	**485.91**	*169.52*	47.02	13	•	•	•
20	17	31	**38.42**	*18.65*	5.17	13	•	•	•

•	•	•	•	•	•	•	•	•	11
1392	1909	2114	1558	2124	2236	1284	1235	7729	2231
1.0	0.8	1.0	1.0	0.8	0.8	1.0	1.0	1.0	1.0
0.2	0.2	0.0	0.0	0.0	0.4	0.0	0.2	0.0	0.2
6	5	5	5	4	6	5	6	5	6
78	53	55	51	45	65	75	61	30	56
125	104	99	131	102	112	129	101	91	103
79	88	95	63	92	98	71	90	94	96
812	542	•	•	•	256	•	549	•	435
897	610	•	•	•	343	•	599	•	502
22	54	•	•	•	39	•	50	•	17

60	11	•	**27.44**	*28.48*	16.44	3
7700	4238	3301	**2657.69**	*2147.76*	536.94	16
1.0	1.0	0.8	**0.95**	*0.09*	0.02	16
0.6	0.0	0.2	**0.13**	*0.18*	0.04	16
8	5	5	**5.38**	*0.89*	0.22	16
66	58	80	**60.83**	*12.98*	3.25	16
106	95	131	**110.37**	*14.85*	3.71	16
93	96	89	**86.14**	*11.23*	2.81	16
461	•	602	**522.43**	*170.13*	64.30	7
521	•	677	**592.67**	*171.18*	64.70	7
63	•	73	**45.43**	*20.66*	7.81	7

Appendix 6c: Participants in study 3: Motor skill acquisition

	Participant	D1	D2	D3	D4	D5
	Age	20	21	21	22	21
	IQ (short-form WAIS-III)	151	110	133	130	113
	Sex	M	F	M	M	F
	Handedness	R	L	L	R	R
WAIS-III subtest age-scaled scores	Vocabulary	17	11	14	11	12
	Similarities	17	10	14	12	13
	Digit Span	9	9	9	8	11
	Picture Completion	15	9	15	15	12
	Digit-Symbol Coding	8	9	10	10	8
	Block Design	16	16	14	18	11
	Brown ADD score	69	39	53	55	52
	NWR time (s)	67	61	88	76	65
	NWR errors	18	9	16	13	8
	WORD spelling (raw score)	35	41	34	37	46
	DAST 1-minute reading	81	89	82	74	103
	DAST 1-minute writing	23	30	29	46	19
	DAST 2-minute spelling	19	31	29	28	27
	Alcohol consumed the day before the experiment (approx. units)	0	2	0	0	0
	Alcohol consumed between days 1 & 2 of the experiment (approx. units)	12	0	0	0	0
	Hours slept between days 1&2 of the experiment	7.5	7.5	8.0	8.0	7.0
	Sequence performed	B	A	B	B	A
	Edinburgh handedness score (part 1)	0.90	-1.00	-1.00	0.87	0.30
	Edinburgh handedness score (part 2)	0.74	-0.43	-0.87	0.87	0.33
	Extent of hand dominance	0.82	0.72	0.94	0.87	0.32
Total correctly performed sequences	Pre-1	3	12	4	8	7
	Pre-2	3	17	6	12	14
	Pre-3	•	13	8	12	12
	Pre-4	0	15	9	11	12
	Post-1	5	19	9	10	13
	Post-2	6	21	8	13	17
	Post-3	4	23	12	15	15
	Post-4	7	25	12	13	17
	24hr-1	9	19	8	15	16
	24hr-2	10	25	10	16	16
	24hr-3	4	24	7	14	19

D6	D7	D8	D9	D10	D11	D12	D13	Mean	St. Dev	St. Err.
22	23	21	22	21	24	21	20	21.46	*1.13*	0.31
126	140	115	103	130	121	121	111	123.38	*13.43*	3.72
M	M	F	F	M	F	M	M			
R	R	R	L	R	L	R	R			
11	14	12	12	13	15	13	11	12.77	*1.83*	0.51
15	17	15	12	18	15	13	13	14.15	*2.30*	0.64
10	15	7	7	10	10	11	9	9.62	*2.06*	0.57
13	13	12	12	13	9	15	11	12.62	*2.10*	0.58
9	9	8	10	8	12	7	9	9.00	*1.29*	0.36
15	16	10	6	12	13	11	12	13.08	*3.23*	0.89
98	51	42	46	20	53	76	48	54.00	*18.90*	5.24
90	98	86	58	67	78	82	121	79.77	*17.41*	4.83
15	10	11	17	9	11	16	6	12.23	*3.85*	1.07
35	38	39	38	40	46	37	38	38.77	*3.77*	1.04
65	108	84	109	95	87	83	80	87.69	*13.01*	3.61
30	29	33	34	31	31	33	29	30.54	*6.20*	1.72
28	27	32	29	32	35	31	29	29.00	*3.79*	1.05
2	0	0	0	0	0	16	0	1.54	*4.41*	1.22
13	11	0	0	0	0	0	0	2.77	*5.28*	1.46
5.0	8.0	9.0	10.0	8.5	8.0	7.0	9.0	7.88	*1.21*	0.34
B	A	B	A	A	A	B	B			
0.76	0.75	1.00	-1.00	0.73	0.17	0.58	1.00	0.31	*0.79*	0.22
0.49	0.52	0.95	-0.89	0.67	0.15	0.62	0.71	0.30	*0.63*	0.17
0.63	0.64	0.98	0.95	0.70	0.16	0.60	0.86	0.70	*0.24*	0.07
9	9	5	1	3	3	7	10	6.23	*3.32*	0.92
12	20	6	2	5	11	8	18	10.31	*5.85*	1.62
11	20	9	3	5	•	8	21	11.09	*5.56*	1.68
13	23	10	7	7	10	12	16	11.15	*5.40*	1.50
13	20	13	11	13	19	11	29	14.23	*6.15*	1.71
14	23	15	5	14	18	14	26	14.92	*6.26*	1.74
15	19	14	8	14	19	13	25	15.08	*5.63*	1.56
11	23	16	6	12	16	15	28	15.46	*6.58*	1.82
16	19	17	9	13	20	13	14	14.46	*3.97*	1.10
16	20	16	9	11	16	16	20	15.46	*4.61*	1.28
19	23	13	9	8	18	15	20	14.85	*6.36*	1.76

n	C1	C2	C3	C4	C5	C6	C7	C8	C9	C10
13	24	24	22	23	22	21	20	20	24	24
13	130	121	133	113	111	130	108	101	135	145
	M	M	F	M	M	M	F	F	F	M
	R	L	R	L	R	R	R	R	R	R
13	15	15	11	18	14	13	17	12	13	17
13	14	14	16	11	13	14	12	10	17	15
13	13	8	17	11	12	10	12	9	14	17
13	12	9	12	8	9	13	7	8	13	15
13	12	9	13	14	11	14	7	8	14	16
13	15	14	18	11	11	16	9	11	15	15
13	53	36	48	57	15	18	38	14	10	32
13	50	58	34	44	43	42	48	42	38	36
13	6	7	0	7	4	6	1	4	2	1
13	46	48	48	45	47	47	44	48	47	46
13	133	109	118	124	119	121	94	119	•	125
13	34	34	36	40	33	43	33	33	•	34
13	40	39	40	35	37	38	31	40	•	36
13	2	0	0	0	0	16	0	0	0	3
13	7	0	0	2	0	0	0	0	0	7
13	7.0	7.0	6.5	5.0	7.0	8.0	8.0	7.0	8.0	6.0
	B	B	A	A	A	A	B	A	A	B
13	1.00	-0.76	0.95	-1.00	0.75	0.63	1.00	1.00	0.90	1.00
13	0.96	-0.96	0.88	-0.67	0.73	0.52	0.72	0.89	0.82	0.69
13	0.98	0.86	0.92	0.84	0.74	0.58	0.86	0.95	0.86	0.85
13	9	10	9	11	11	7	11	6	13	11
13	12	13	11	13	15	16	15	5	15	4
11	9	17	12	10	18	18	15	7	15	4
13	13	10	12	13	21	20	13	5	12	13
13	18	20	16	21	19	17	12	13	18	20
13	21	19	19	12	21	•	15	14	19	20
13	19	20	17	18	25	19	12	13	19	20
13	23	19	16	13	23	13	15	16	21	20
13	24	17	15	15	21	19	20	11	22	21
13	22	18	16	12	24	15	21	8	22	25
13	22	20	16	10	23	19	22	7	19	22

C11	C12	Mean	*St. Dev*	St. Err.	n
22	21	22.25	*1.54*	0.45	12
146	143	126.33	*15.31*	4.42	12
F	M				
R	R				
17	17	14.92	*2.31*	0.67	12
14	18	14.00	*2.34*	0.67	12
13	13	12.42	*2.78*	0.80	12
18	9	11.08	*3.32*	0.96	12
12	8	11.50	*2.91*	0.84	12
14	17	13.83	*2.76*	0.80	12
50	65	36.33	*18.72*	5.40	12
70	40	45.42	*10.11*	2.92	12
3	4	3.75	*2.42*	0.70	12
46	49	46.75	*1.42*	0.41	12
87	128	116.09	*14.14*	4.26	11
33	38	35.55	*3.39*	1.02	11
40	37	37.55	*2.81*	0.85	11
0	0	1.75	*4.59*	1.33	12
0	5	1.75	*2.86*	0.83	12
8.0	3.0	6.71	*1.48*	0.43	12
A	B				
0.80	1.00	0.61	*0.71*	0.20	12
0.86	0.79	0.52	*0.64*	0.18	12
0.83	0.90	0.85	*0.10*	0.03	12
10	2	9.17	*2.95*	0.85	12
11	8	11.50	*3.97*	1.14	12
10	10	12.08	*4.52*	1.31	12
15	14	13.42	*4.17*	1.20	12
18	13	17.08	*3.00*	0.87	12
16	16	17.45	*3.01*	0.91	11
19	16	18.08	*3.40*	0.98	12
17	17	17.75	*3.47*	1.00	12
16	15	18.00	*3.77*	1.09	12
18	14	17.92	*5.14*	1.48	12
15	17	17.67	*5.02*	1.45	12

	24hr-4	14	23	9	17	20
Mean time taken to perform a complete sequence (s)	Pre-1	3.27	1.44	4.73	2.28	2.79
	Pre-2	2.90	1.37	3.88	1.66	1.82
	Pre-3	•	1.30	3.05	1.38	2.03
	Pre-4	•	1.16	2.89	1.63	1.80
	Post-1	1.48	1.07	2.04	1.36	1.52
	Post-2	1.44	1.05	2.02	1.30	1.47
	Post-3	2.08	1.03	1.86	1.39	1.39
	Post-4	1.70	0.99	1.95	1.44	1.36
	24hr-1	1.55	1.16	2.23	1.32	1.48
	24hr-2	1.47	1.01	2.10	1.23	1.47
	24hr-3	1.57	1.03	2.11	1.21	1.41
	24hr-4	1.46	1.01	2.01	1.23	1.25
Simple errors (deviations from correctly performed sequences)	Pre-1	1	6	0	2	0
	Pre-2	1	5	0	7	0
	Pre-3	•	7	0	5	1
	Pre-4	0	8	0	5	1
	Post-1	3	4	2	8	5
	Post-2	3	5	3	8	1
	Post-3	4	2	1	2	1
	Post-4	6	2	1	5	3
	24hr-1	6	3	2	3	3
	24hr-2	4	1	2	7	2
	24hr-3	4	4	4	8	0
	24hr-4	3	4	2	9	0
Raw speed (number of contacts per 30s trial)	Pre-1	78	189	50	115	96
	Pre-2	82	205	62	164	148
	Pre-3	•	219	86	182	132
	Pre-4	88	248	92	159	150
	Post-1	147	237	112	189	168
	Post-2	140	250	114	200	183
	Post-3	120	250	136	189	178
	Post-4	142	275	134	185	191
	24hr-1	153	217	118	197	186
	24hr-2	157	266	120	221	180
	24hr-3	146	256	110	225	191
	24hr-4	181	264	136	228	212
Training	Number of correctly performed sequences	44	78	75	38	132
	Time taken per sequence (s)	2.24	1.37	2.27	1.90	1.62
	Simple errors	20	49	23	21	28
	Raw speed	1315	1517	1391	1220	1664
Overnght improvement	Total completed sequences (effect sizes)	0.56	-2.01	-1.37	0.56	-0.40
	Simple errors (effect sizes)	-0.70	-1.02	-1.02	-0.05	-0.70
	Total sequences and simple errors - combined effect sizes	-0.14	-3.03	-2.39	0.51	-1.10

17	21	16	8	12	15	16	19	15.92	*4.42*	1.23
2.24	2.21	3.58	2.80	5.31	2.19	2.82	1.87	**2.89**	*1.11*	0.31
1.84	1.26	2.74	3.63	3.45	1.70	2.21	1.34	**2.29**	*0.92*	0.26
1.69	1.20	2.47	3.32	3.34	•	2.22	1.08	**2.10**	*0.85*	0.26
1.82	1.18	2.34	2.84	3.08	1.66	1.98	1.01	**1.95**	*0.70*	0.20
1.43	1.08	1.63	2.11	2.00	1.23	1.62	0.92	**1.50**	*0.38*	0.11
1.68	1.01	1.56	1.84	1.73	1.16	1.75	0.95	**1.46**	*0.34*	0.10
1.46	1.11	1.53	1.90	1.84	1.13	1.57	0.96	**1.48**	*0.36*	0.10
1.56	1.05	1.44	1.64	2.11	1.14	1.59	0.90	**1.45**	*0.37*	0.10
1.48	1.10	1.49	2.08	2.13	1.18	1.54	1.17	**1.53**	*0.39*	0.11
1.41	1.06	1.42	2.19	2.19	1.16	1.49	1.11	**1.49**	*0.42*	0.12
1.43	1.08	1.40	2.33	2.59	1.04	1.54	1.02	**1.52**	*0.52*	0.14
1.51	1.05	1.42	2.23	2.15	1.18	1.53	0.99	**1.46**	*0.42*	0.12
1	4	2	1	0	1	1	4	**1.77**	*1.83*	0.51
2	1	3	2	2	2	2	1	**2.15**	*1.95*	0.54
2	3	3	2	2	•	1	2	**2.55**	*1.97*	0.59
1	1	3	4	1	3	2	6	**2.69**	*2.46*	0.68
5	3	3	1	1	2	3	0	**3.08**	*2.10*	0.58
2	3	1	2	1	3	1	2	**2.69**	*1.97*	0.55
4	5	3	3	1	4	3	2	**2.69**	*1.32*	0.36
5	2	1	4	2	4	3	2	**3.08**	*1.61*	0.45
2	3	2	3	0	3	2	14	**3.54**	*3.41*	0.94
4	2	1	4	1	4	1	4	**2.85**	*1.82*	0.50
0	2	5	2	2	4	1	6	**3.23**	*2.35*	0.65
1	4	3	3	0	5	1	7	**3.23**	*2.65*	0.74
115	133	74	85	48	125	92	140	**103.08**	*38.98*	10.81
144	218	100	88	70	154	109	192	**133.54**	*52.39*	14.53
150	227	122	105	79	•	117	239	**150.73**	*57.44*	17.32
145	234	120	112	90	156	134	246	**151.85**	*57.29*	15.89
192	256	156	136	137	207	152	298	**183.62**	*53.80*	14.92
162	272	166	155	153	248	155	280	**190.62**	*54.30*	15.06
182	254	170	159	154	256	169	269	**191.23**	*49.49*	13.73
166	264	184	166	132	245	169	298	**196.23**	*55.79*	15.47
189	246	180	139	132	241	165	228	**183.92**	*41.57*	11.53
192	261	186	124	126	238	172	236	**190.69**	*50.89*	14.11
195	252	186	127	99	264	164	254	**189.92**	*57.95*	16.07
184	263	190	129	128	232	177	273	**199.77**	*50.75*	14.08
86	137	56	26	69	124	89	141	**84.23**	*38.94*	10.80
1.81	1.59	2.18	2.41	2.52	1.47	2.02	1.28	**1.90**	*0.41*	0.11
28	26	33	22	23	29	44	21	**28.23**	*9.01*	2.50
1469	1615	1252	2094	907	1671	1517	1599	**1479.31**	*283.67*	78.68
1.53	-1.37	0.24	0.88	0.24	1.21	-0.72	-4.59	**-0.40**	*1.65*	0.46
0.27	-1.02	-1.02	-0.38	-0.05	-0.38	-0.38	-4.57	**-0.85**	*1.20*	0.33
1.80	-2.39	-0.78	0.51	0.19	0.83	-1.10	-9.16	**-1.25**	*2.76*	0.77

13	23	20	19	16	23	18	22	10	24	28
13	2.62	2.21	2.78	1.46	2.33	2.53	2.12	3.37	1.63	1.21
13	1.64	1.90	2.39	1.22	1.69	1.62	1.66	3.07	1.65	1.27
11	1.50	1.61	2.11	1.15	1.44	1.40	1.56	2.87	1.50	1.16
12	1.37	1.64	2.08	1.06	1.22	1.22	1.49	2.59	1.43	1.17
13	1.13	1.28	1.47	0.96	1.01	1.07	1.52	1.94	1.12	0.99
13	1.16	1.29	1.33	1.01	1.04	•	1.30	1.83	1.03	0.98
13	1.21	1.41	1.26	0.95	1.01	1.15	1.42	1.64	1.06	0.98
13	1.07	1.40	1.34	0.95	1.00	1.25	1.19	1.60	1.12	0.95
13	1.11	1.59	1.58	1.03	1.20	1.11	1.21	2.20	1.19	0.98
13	1.12	1.50	1.54	1.00	1.09	0.99	1.27	1.75	1.14	0.97
13	1.08	1.38	1.49	0.95	1.09	0.99	1.23	1.88	1.17	0.97
13	1.07	1.34	1.38	0.97	1.06	0.97	1.15	1.74	1.05	0.93
13	2	1	0	4	0	1	1	1	4	8
13	4	0	1	6	0	0	1	4	2	4
11	7	0	0	4	0	1	3	2	3	3
13	6	2	0	5	0	1	3	1	5	8
13	3	1	2	4	4	6	5	0	6	6
13	2	2	6	5	4	•	4	0	8	8
13	2	0	8	5	2	3	6	2	5	6
13	2	1	11	4	5	5	6	1	3	5
13	0	0	1	5	0	5	2	0	0	5
13	1	1	1	4	0	7	1	4	2	4
13	4	0	1	5	1	6	0	3	4	5
13	2	1	0	3	4	6	3	4	2	1
13	105	115	95	175	114	84	130	80	174	204
13	169	145	112	207	155	164	163	94	165	184
11	170	174	125	218	185	186	175	96	180	169
13	214	155	124	248	218	208	180	96	194	220
13	229	212	180	247	248	236	178	136	248	251
13	226	215	215	234	250	•	194	145	274	264
13	221	202	212	237	265	230	184	152	261	262
13	244	202	208	241	266	194	208	168	243	275
13	241	177	169	230	223	237	221	118	229	270
13	233	188	174	257	246	257	220	165	234	285
13	246	206	182	249	246	250	227	162	229	269
13	244	209	193	256	260	261	237	156	259	292
13	136	117	125	125	89	126	84	62	121	98
13	1.74	1.71	1.87	1.15	1.45	1.60	1.80	2.30	1.99	1.28
13	29	34	52	29	23	31	50	43	48	21
13	1638	1629	1660	1687	1393	1586	1541	1466	1651	1168
13	0.24	-0.72	-0.40	0.56	-0.72	1.85	1.53	-1.69	0.24	0.24
13	-0.05	-0.38	2.53	-1.02	0.91	-0.70	0.59	-0.38	0.27	-0.70
13	0.19	-1.10	2.13	-0.46	0.19	1.15	2.12	-2.07	0.51	-0.46

18	16	19.75	*4.69*	1.35	12
2.02	2.98	2.27	*0.64*	0.18	12
1.89	2.84	1.90	*0.58*	0.17	12
1.79	2.29	1.70	*0.50*	0.14	12
1.64	1.77	1.56	*0.43*	0.13	12
1.29	1.70	1.29	*0.31*	0.09	12
1.32	1.52	1.26	*0.26*	0.08	11
1.41	1.58	1.26	*0.23*	0.07	12
1.23	1.57	1.22	*0.22*	0.06	12
1.49	1.59	1.36	*0.35*	0.10	12
1.45	1.55	1.28	*0.27*	0.08	12
1.40	1.52	1.26	*0.28*	0.08	12
1.38	1.48	1.21	*0.25*	0.07	12
2	1	2.08	*2.27*	0.66	12
3	0	2.08	*2.07*	0.60	12
2	2	2.25	*2.01*	0.58	12
2	1	2.83	*2.59*	0.75	12
2	2	3.42	*2.07*	0.60	12
4	1	4.00	*2.65*	0.80	11
0	0	3.25	*2.70*	0.78	12
3	0	3.83	*2.95*	0.85	12
1	1	1.67	*2.10*	0.61	12
1	3	2.42	*2.02*	0.58	12
3	1	2.75	*2.09*	0.60	12
2	2	2.50	*1.62*	0.47	12
133	63	122.67	*42.89*	12.38	12
140	96	149.50	*34.38*	9.92	12
157	122	163.08	*33.48*	9.66	12
169	155	181.75	*44.04*	12.71	12
207	154	210.50	*39.91*	11.52	12
202	181	218.18	*37.61*	11.34	11
192	168	215.50	*37.43*	10.81	12
213	170	219.33	*34.67*	10.01	12
181	170	205.50	*42.40*	12.24	12
184	176	218.25	*39.73*	11.47	12
200	187	221.08	*33.17*	9.58	12
192	180	228.25	*41.19*	11.89	12
127	81	107.58	*23.70*	6.84	12
1.68	1.93	1.71	*0.31*	0.09	12
41	48	37.42	*10.92*	3.15	12
1680	1497	1549.67	*151.89*	43.85	12
-0.40	-0.72	0.00	*1.00*	0.29	12
-0.05	-1.02	0.00	*1.00*	0.29	12
-0.46	-1.75	0.00	*1.34*	0.39	12

Total sequences - simple errors combination score	Pre-1	-1.61	-0.76	-0.84	-0.36	0.18
	Pre-2	-1.62	-0.03	-0.38	-2.25	1.64
	Pre-3	•	-2.17	0.22	-1.39	0.60
	Pre-4	-2.13	-1.62	0.03	-1.42	0.37
	Post-1	-3.83	0.36	-2.01	-4.58	-2.13
	Post-2	-3.42	0.80	-2.76	-2.99	0.98
	Post-3	-4.42	1.91	-0.96	-0.44	-0.07
	Post-4	-3.84	2.71	-0.70	-1.77	0.07
	24hr-1	-4.45	-0.37	-2.81	-1.43	-1.16
	24hr-2	-2.32	2.08	-1.33	-2.64	-0.17
	24hr-3	-3.32	0.67	-2.72	-3.24	1.58
	24hr-4	-1.53	-0.23	-1.98	-4.59	1.59
Raw speed - simple errors combination score	Pre-1	-0.57	-0.18	-0.78	-0.14	0.29
	Pre-2	-1.44	0.20	-1.54	-1.96	0.97
	Pre-3	•	-0.70	-1.18	-0.81	-0.31
	Pre-4	-1.03	-0.49	-0.94	-1.35	-0.01
	Post-1	-1.39	0.38	-1.78	-2.76	-1.83
	Post-2	-1.70	0.47	-2.39	-2.00	0.20
	Post-3	-2.83	1.38	-1.29	-0.25	-0.17
	Post-4	-2.97	2.23	-1.50	-1.39	-0.53
	24hr-1	-3.30	-0.36	-2.22	-0.83	-1.09
	24hr-2	-2.33	1.90	-2.27	-2.20	-0.76
	24hr-3	-2.86	0.46	-3.95	-2.39	0.41
	24hr-4	-1.46	-0.06	-1.93	-4.01	1.15

0.42	-0.90	-1.38	-2.29	-1.18	-1.61	-0.26	-0.56	**-0.86**	*0.76*	0.21
0.17	2.67	-1.83	-2.36	-1.60	-0.09	-0.84	2.16	**-0.33**	*1.66*	0.46
-0.11	1.38	-1.06	-1.88	-1.44	•	-0.28	2.10	**-0.37**	*1.37*	0.41
0.61	3.01	-0.88	-1.99	-0.83	-0.88	-0.02	-0.60	**-0.49**	*1.36*	0.38
-2.13	1.17	-1.16	-0.86	-0.19	1.33	-1.83	5.63	**-0.79**	*2.60*	0.72
-0.39	2.22	0.32	-3.38	-0.01	0.56	-0.01	3.59	**-0.35**	*2.20*	0.61
-1.19	-0.38	-1.11	-2.88	-0.37	-0.01	-1.40	2.50	**-0.68**	*1.77*	0.49
-2.34	2.14	0.46	-3.45	-1.04	-0.56	-0.51	3.58	**-0.40**	*2.24*	0.62
-0.69	-0.37	-0.42	-3.02	-0.54	-0.10	-1.49	-6.93	**-1.83**	*2.00*	0.56
-1.16	0.61	0.33	-2.52	-0.64	-1.16	0.33	-0.38	**-0.69**	*1.37*	0.38
1.58	1.42	-2.00	-1.37	-1.57	-0.53	0.30	-1.09	**-0.79**	*1.78*	0.49
0.34	-0.66	-1.11	-2.81	-0.11	-2.55	0.12	-2.93	**-1.27**	*1.68*	0.47
0.30	-0.60	-1.10	-0.40	-0.82	0.53	-0.24	-0.44	**-0.32**	*0.48*	0.13
-0.12	2.52	-1.88	-1.75	-2.27	0.17	-1.14	1.76	**-0.50**	*1.54*	0.43
-0.27	1.54	-1.60	-1.61	-2.39	•	-0.75	2.39	**-0.52**	*1.39*	0.42
-0.13	1.89	-1.47	-2.03	-1.38	-0.65	-0.76	0.24	**-0.62**	*0.99*	0.28
-1.23	1.34	-1.16	-0.70	-0.67	0.60	-1.26	3.85	**-0.51**	*1.71*	0.48
-0.74	1.81	-0.25	-0.92	-0.60	1.17	-0.55	2.40	**-0.24**	*1.43*	0.40
-1.17	0.38	-1.12	-1.42	-0.81	0.80	-1.15	1.89	**-0.44**	*1.30*	0.36
-1.93	1.91	-0.06	-1.59	-1.90	0.68	-1.17	2.89	**-0.41**	*1.82*	0.50
-0.55	0.32	-0.76	-2.20	-0.94	0.20	-1.11	-5.33	**-1.40**	*1.55*	0.43
-1.44	1.28	-0.11	-3.16	-1.62	-0.29	-0.46	-0.34	**-0.91**	*1.46*	0.41
0.53	1.29	-2.13	-2.48	-3.32	0.70	-0.89	-0.56	**-1.17**	*1.76*	0.49
-0.15	-0.08	-1.24	-2.72	-0.89	-1.45	-0.32	-1.68	**-1.14**	*1.33*	0.37

13	-0.02	0.76	0.86	-0.22	1.54	-0.26	1.10	-0.60	0.46	-1.98
13	-0.80	1.39	0.40	-1.52	1.89	2.14	1.41	-2.57	0.92	-2.82
11	-3.05	2.21	1.10	-1.33	2.43	1.93	0.27	-1.00	0.27	-2.16
13	-1.32	-0.50	0.75	-0.94	2.92	2.29	-0.16	-1.31	-1.18	-2.10
13	0.51	2.14	0.32	1.02	0.36	-1.28	-2.46	0.29	-0.95	-0.28
13	1.93	1.27	-0.24	-2.19	1.18	•	-0.81	0.36	-1.00	-0.67
13	0.73	1.77	-2.08	-0.67	2.50	0.36	-2.81	-1.03	-0.38	-0.45
13	2.14	1.32	-2.93	-1.43	1.12	-1.77	-1.53	0.46	1.22	0.25
13	2.39	0.53	-0.48	-2.38	1.59	-1.32	0.37	-1.07	1.85	-0.79
13	1.50	0.72	0.33	-1.93	2.38	-2.84	1.30	-2.71	1.00	0.59
13	0.27	1.78	0.50	-2.60	1.90	-1.29	2.18	-2.25	-0.33	-0.21
13	1.00	0.98	1.38	-1.11	-0.23	-2.53	0.17	-3.00	1.21	2.68
13	-0.38	0.30	0.27	0.38	0.71	-0.43	0.65	-0.52	0.35	-0.70
13	-0.36	0.88	-0.57	-0.22	1.17	1.43	0.92	-2.54	0.49	0.08
11	-2.16	1.45	-0.02	0.77	1.78	1.31	-0.02	-1.88	0.13	-0.20
13	-0.49	-0.29	-0.22	0.67	1.92	1.30	-0.10	-1.24	-0.56	-1.13
13	0.67	1.21	-0.08	0.63	0.66	-0.61	-1.58	-0.21	-0.31	-0.24
13	0.96	0.67	-0.84	0.04	0.85	•	-0.64	-0.43	-0.03	-0.29
13	0.61	0.84	-1.85	-0.07	1.79	0.48	-1.86	-1.23	0.57	0.22
13	1.33	0.46	-2.76	0.57	0.95	-1.13	-1.06	-0.52	0.97	1.21
13	1.63	0.12	-0.54	-1.01	1.21	-0.84	0.21	-1.27	1.35	-0.06
13	1.07	-0.06	-0.41	0.19	1.89	-1.29	0.75	-2.12	0.60	0.90
13	0.15	0.86	-0.34	-0.23	1.59	-0.68	1.49	-1.90	-0.36	0.37
13	0.69	0.46	0.68	0.37	-0.15	-1.36	-0.10	-2.68	1.05	2.47

0.32	-1.95	0.00	*1.10*	0.32	12
-0.57	0.13	0.00	*1.68*	0.48	12
-0.34	-0.34	0.00	*1.73*	0.50	12
0.70	0.85	0.00	*1.54*	0.44	12
0.99	-0.68	0.00	*1.22*	0.35	12
-0.48	0.65	0.00	*1.20*	0.36	11
1.47	0.59	0.00	*1.56*	0.45	12
0.07	1.08	0.00	*1.55*	0.45	12
-0.21	-0.48	0.00	*1.41*	0.41	12
0.72	-1.05	0.00	*1.71*	0.49	12
-0.65	0.70	0.00	*1.55*	0.45	12
-0.06	-0.49	0.00	*1.64*	0.47	12
0.28	-0.91	0.00	*0.55*	0.16	12
-0.72	-0.55	0.00	*1.09*	0.31	12
-0.06	-1.10	0.00	*1.24*	0.36	12
0.03	0.10	0.00	*0.92*	0.27	12
0.60	-0.73	0.00	*0.78*	0.23	12
-0.43	0.15	0.00	*0.61*	0.18	11
0.58	-0.07	0.00	*1.11*	0.32	12
0.10	-0.12	0.00	*1.20*	0.35	12
-0.26	-0.52	0.00	*0.95*	0.27	12
-0.16	-1.35	0.00	*1.16*	0.33	12
-0.75	-0.19	0.00	*0.98*	0.28	12
-0.57	-0.86	0.00	*1.30*	0.38	12