Children with dyslexia acquire skill more slowly[1]

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Abstract

Two studies are reported in which a group of adolescent children with dyslexia and a group of normal children matched for age and IQ undertook extended training. In Study 1, which comprised three phases of learning over an 18 month period, the children learned to navigate via key presses around a fixed circuit of a computer maze. It was concluded that, following extended training under these optimal conditions, the children with dyslexia had normal 'strength' of automatisation (as assessed by resistance to unlearning, by ease of relearning, and by dual task performance) but that their 'quality' of automatisation (as assessed by speed and accuracy) was impaired. Study 2 investigated the blending of two compatible simple reaction responses into a two choice reaction. Although performance on the simple reactions was equivalent across groups, the children with dyslexia had more difficulty combining the two skills at first and showed significantly less learning over the course of the training period. The estimated learning rate was around 50% slower for the children with dyslexia, leading to the prediction that the proportionate slowing in acquisition time would increase as the square root of the normal acquisition time. A connectionist framework may provide a natural explanation of the phenomena.

Introduction

Specific developmental dyslexia, or dyslexia for short, is formally defined as "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" (from the definition by the World Federation of Neurologists, 1968). In other words, children of normal or above normal intelligence who, for some otherwise inexplicable reason, have severe problems learning to read and spell.

One of the fascinations of dyslexia for researchers is that, whatever one's interest in human behaviour and performance, children with dyslexia will obligingly show interestingly abnormal behaviour in precisely that behaviour. Researchers interested in reading and in language formulated a general 'language deficit' hypothesis (Vellutino, 1979) which has been refined over the years (Miles, 1983; Snowling et al, 1986; Stanovich, 1988) to provide what is arguably the consensus theoretical belief of most psychology researchers, namely that children with dyslexia suffer from an early impairment in their phonological skills, and this impairment prevents them from acquiring the word decoding and blending skills necessary for normal acquisition of the skill of reading. By contrast, however, many American researchers have studied the biological substrate. Again, dyslexia has provided intriguing abnormalities. Large scale twin and familial studies (e.g., Smith et al, 1983) has established specific abnormalities both of chromosome 15 and, more recently, chromosome 6 (Lubs et al, 1991). Studies of brain electrical activity in response to different types of stimulus have shown abnormalities for the processing of linguistic stimuli (Duffy et al, 1980; Hynd et al, 1990). Most directly, comparative neuroanatomical studies of dyslexic brains have established "*a uniform absence of left-right asymmetry in the language area and focal dysgenesis referrable to midgestation ... possibly having widespread cytoarchitectonic and connectional repercussions. ... Both types of changes in the male brains are associated with increased numbers of neurons and connections and qualitatively different patterns of cellular architecture and connections*" (Galaburda, Rosen and Sherman, 1989, p383). One significant recent development has been the re-establishment of visual deficits, in this case in rapid visual processing, specifically the threshold for the detection of flicker (Lovegrove et al, 1990), and in an interdisciplinary project involving both psychophysics and neuroanatomical analysis, this deficit has been linked to neuroanatomical abnormalities in the magnocellular pathway linking the eye to the visual cortex via the lateral geniculate nucleus (Livingstone et al, 1991).

There is, therefore, a wealth of research evidence about abnormalities in dyslexia. Unfortunately, though, many of the frameworks adopted for dyslexia research have difficulty in accommodating the results

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obtained from the other disciplines. The objective of our research has been to find a sufficiently broad framework, and the framework we investigated was that of learning.

It is evident that a learning perspective should be of use in analyses of problems in learning to read. Furthermore, learning is one of the major frameworks for cognitive science research, with both connectionist and symbolic architectures such as Soar and ACT* assigning a central role to learning. Paradoxically, however, learning is just about the only framework not used in previous dyslexia research. The reason that theorists have not seriously considered learning as a viable framework is that it fails to explain the apparent specificity of the deficits in dyslexia. If they have a general problem in learning, why do children with dyslexia not show problems in *all* skills, cognitive and motor? In our approach to this difficulty we were encouraged first by the observation that, whatever skill theorists had examined carefully (with the single exception of spatial skills), a deficit had been observed in children with dyslexia. Furthermore, careful observation of children with dyslexia suggests that, although they appear to be behaving normally, they show unusual lapses of concentration and get tired more quickly than normal when performing a skill (Augur, 1985). In the words of the parent of one of our panel of children with dyslexia, it might be that life for a child with dyslexia might be like living in a foreign country, where it is possible to get by adequately, but only at the expense of continual concentration and effort. This belief in a learning deficit led us (Nicolson and Fawcett, 1990) to formulate and test two linked hypotheses: first, the Dyslexic Automatisation Deficit (DAD) hypothesis, that children with dyslexia have unusual difficulty in automatising any skill, whether motor or cognitive, and second, the 'Conscious Compensation' hypothesis, namely that children with dyslexia are normally able to overcome their automatisation deficit by means of consciously compensating for it, that is, by trying harder and/or by using strategies to minimise or mask the deficit. These hypotheses were supported by studies showing hitherto unexpected deficits in balance under dual task conditions (Nicolson and Fawcett, 1990; Fawcett and Nicolson, 1992).

The balance studies are necessarily indirect investigations of learning, and the only way to provide direct evidence for or against DAD was to undertake a long-term training study on a novel skill, and to attempt to identify in which stage, if any, the children with dyslexia showed impaired performance and consequently we undertook two extended studies designed to investigate the microstructure of learning in normal and children with dyslexia. Before discussing the experiments individually, it is important to state the basis for our selection of subjects. In brief, we wanted to study 'pure' dyslexia, uncontaminated by factors such as low IQ, economic disadvantage and so on. Consequently, we used the standard exclusionary criterion of 'children of normal or above normal IQ (operationalised as IQ of 90 or more on the Wechsler Intelligence Scale for Children), without known primary emotional or behavioural or socioeconomic problems, whose reading age (RA) was at least 18 months behind their chronological age (CA).

Our subjects in Study 1 were 13 children with dyslexia (mean chronological age 14.9, range 13.5 to 15.6; mean IQ 108.8, range 90 to 140; mean reading age 11.9, range 8.1 to 14.4) and 8 normal children (mean age 15.1 years, range 14.5 to 15.9; mean IQ 109.5, range 92 to 130; reading ages around ceiling [15.0] on the Schonell test used), with groups matched overall for age and IQ. The same groups of children participated in Study 2, though by then they were around 1.5 years older.

Study 1: Extended Training on a computer maze

The first study involved learning of a complex eye-hand coordination skill, typical of everyday activity of many teenage children, namely performance on an arcade-type computer game, specially redesigned to allow performance speed and accuracy to be monitored continuously. The subjects had to navigate a 'Pacman' icon round a fixed track of computer mazeusing specified key presses to move left, right, up and,down. The critical question was how the skill developed with practice, and so extended training was given over a period of about 6 months until each subject appeared to have stopped improving. Performance was monitored three times per session at 20 minute intervals, with the intervening time taken up with free play on the full game, together with various other tests as part of our testing program. Following a two week respite from the task, the key--movement mappings were then changed incompatibly, thereby forcing the subjects to unlearn their initial finger--movement pairings and to relearn the new pairings, and performance was again monitored until no noticeable improvement was taking place. Finally, one year later, the task was administered again, this time with eight fixed sessions of three trials, in order to examine the amount of forgetting of the skill over one year. Furthermore, various perturbations to the standard procedure were made on the later sessions, in order to examine the susceptibility of the skill to interference. Full details are presented in Nicolson and Fawcett (1992).

The results for the completion times for the first two phases are shown in Figure 1. The children with dyslexia were initially very much slower than the controls, and even after extended training they were still significantly slower. On the other hand, they showed good improvement in speed with practice, and also showed equivalent interference to the controls when the

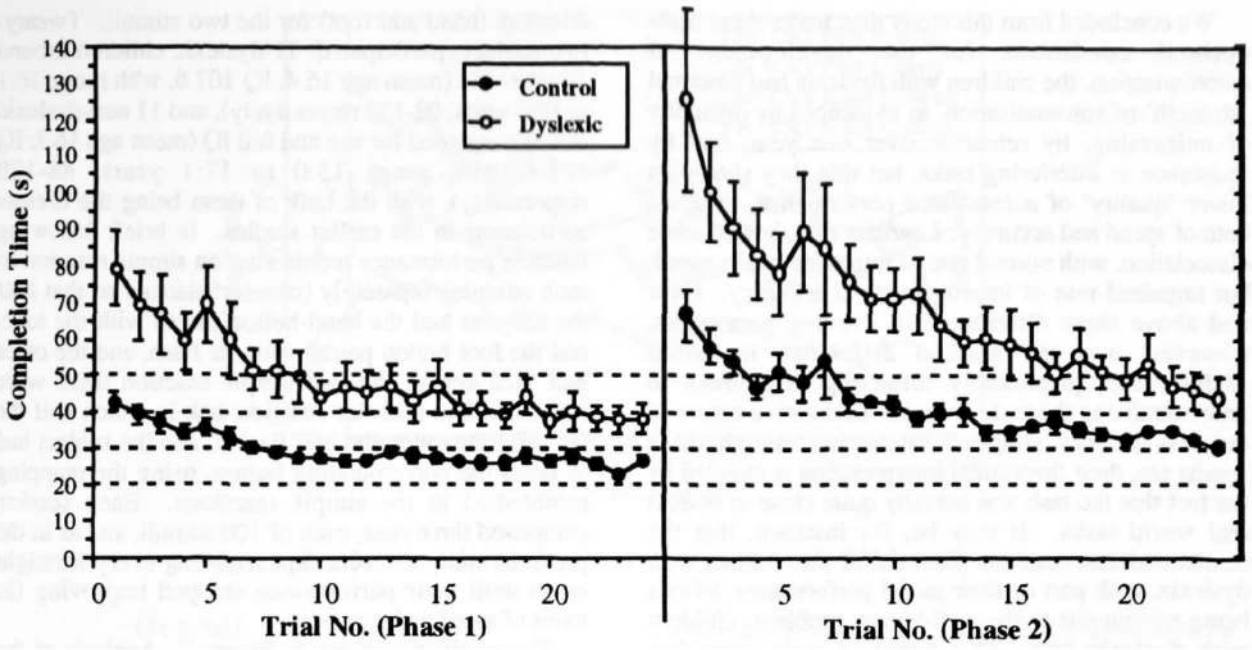

Figure 1. Completion Times in Phases 1 and 2 of the Pacman Training
(error bars are the standard errors)

key mappings were changed. Similar results were obtained for errors (incorrect key presses), with the children with dyslexia making significantly more errors initially and after training, but in contrast to their completion times, and in contrast to the control children, the children with dyslexia showed little reduction in error rate with training.

In order to obtain more accurate estimates of the learning rates for completion times and errors, the group data were fitted using a power law parametric technique which has been established as appropriate for fitting human data on practice (Newell and Rosenbloom, 1981). In brief, the curve fitted is of the form $P(n) = A + Bn^{-\alpha}$ where $P(n)$ refers to performance on trial n, A is the asymptotic performance as $n \rightarrow \infty$, B is a scaling parameter linked directly to initial performance, and α is the learning rate. The best fit was derived by a least squares technique, and the parameters are shown in Table 1.

It may be seen that in neither Phase 1 nor 2 was there a significant difference in learning rate, and that the major difference for learning rate was the parameter B, initial performance. By contrast, the learning rate for error elimination was markedly lower for the children with dyslexia, to the extent that the model predicts that the children with dyslexia would be making more errors after 10,000 trials than the normal children after 100 trials! Performance in phase 3 (one year post-training) indicated that both groups showed good skill retention over the intervening year, and also good relearning, in that after only two sessions of three trials both groups were performing around the level of their previous best performance. Furthermore, the children with dyslexia appeared to be able to cope as well as the controls with changes of layout, with presentation of white noise, and with the need to undertake an auditory detection task while navigating round the maze.

Table 1. Learning Parameters Estimated for Phases 1 and 2 using the Power Law model $T(n)=A+Bn^{-\alpha}$

	Phase 1		Phase 2	
	Control	Dys	Control	Dys
Times				
A	20	20	20	20
B	33.71	91.98	77.96	176.25
α	0.586	0.51	0.57	0.575
r^2	0.807	0.881	0.859	0.890
T(100)	22.3	28.8	25.6	32.5
T(1,000)	20.6	22.7	21.5	23.3
T(10,000)	20.2	20.8	20.4	20.9
Errors				
A	0	0	0	0
B	21.90	26.06	25.64	38.65
α	0.221	0.111	0.2	0.208
r^2	0.693	0.582	0.525	0.799
E(100)	7.9	15.6	10.2	14.8
E(1,000)	4.8	12.1	6.4	9.2
E(10,000)	2.9	9.4	4.1	5.7

We concluded from this study that, under these near-optimal conditions for the development of automatisation, the children with dyslexia had a normal 'strength' of automatisation, as evidenced by difficulty of unlearning, by retention over one year, and by resistance to interfering tasks, but that they showed a lower 'quality' of automatised performance, in terms both of speed and accuracy. Learning rate showed some dissociation, with normal rate of improvement in speed, but impaired rate of improvement in accuracy. Over and above these differences in learning parameters, however, was the marked difference in initial performance, presumably reflecting difficulties in proceduralising the task, since the declarative nature of the task is very simple. Interesting though these results are, their theoretical interpretation is clouded by the fact that the task was actually quite close to several real world tasks. It may be, for instance, that the results underestimate the potential of the children with dyslexia, with part of their initial performance deficits being attributable to the well-known problems children with dyslexia have discriminating right from left (Miles, 1983), or maybe to a comparative lack of prior practice on some of the component skills. On the other hand, it may be that these results overestimate the learning potential of the children with dyslexia. Perhaps their near-normal rate of improvement in speed is partly attributable to the fact that they had much more room for improvement than the control children. These issues could only be resolved by a further experiment, one in which we attempted to ensure that the children with dyslexia had no impairment on the component skills underlying the task to be learned.

Study 2: Extended CRT training

In related research (Nicolson and Fawcett, 1993a), we had established that this group of children with dyslexia had normal speed of simple reaction (that is, pressing a button as soon as they heard a high tone), but that their speed of choice reaction was impaired compared with same age controls. Interestingly, this impairment obtained even for a selective choice reaction, in which the target tone and the response was identical to that of the simple reaction, but an alternative low tone (for which the subject had to make no response) was presented on half the trials. With the intention of further probing this intriguing dissociation, while studying the time course of the automatisation process for a primitive skill, we conducted a further long-term training study, in which we examined the time course of development of choice reaction speed. In order to avoid any problems of left-right confusions or of stimulus discriminability, we used two stimuli of different modalities (tone and flash) and different effectors (hand and foot) for the two stimuli. Twenty-two subjects participated: 11 dyslexic children around 16 years old (mean age 16.4, IQ 107.6, with range 16:1 to 17:0 years, 92-130 respectively), and 11 non-dyslexic children matched for age and full IQ (mean age 16.3, IQ 105.4, with range 15:0 to 17:1 years, 88-128 respectively), with the bulk of them being the tireless participants in the earlier studies. In brief, following baseline performance monitoring on simple reaction to each stimulus separately (counterbalanced so that half the subjects had the hand-button paired with the tone, and the foot-button paired with the flash, and the other half vice versa), the two simple reaction tasks were combined into a choice reaction task in which half the stimuli were tones and half flashes, and the subject had to press the corresponding button, using the mapping established in the simple reactions. Each session comprised three runs, each of 100 stimuli, and as in the previous study subjects kept returning every fortnight or so until their performance stopped improving (in terms of speed and accuracy).

The results are shown in Figure 2. Analysis of the simple reaction performance indicated that there were no significant differences between the groups either for foot or hand, tone or flash. By contrast, initial performance on the choice reaction was significantly slower, and final performance was both significantly slower and less accurate for the children with dyslexia. A parametric learning rate analysis was then performed using the power law equation outlined above. The best fit curves for hand responses were $CRT = 53.9\ n^{-0.073}$ for the children with dyslexia and $CRT = 39.4\ n^{-0.141}$ for the controls. For the foot responses the corresponding best fit curves were $CRT = 62.3\ n^{-0.086}$; $CRT = 50.4\ n^{-0.116}$ respectively. As in the Pacman experiment, the parameter B was higher for the children with dyslexia than the controls (around 30% on average). This is a particularly interesting result in view of the near-equality of the baseline simple reaction performance on which the CRT was founded. Even more interesting, however, is the difference in learning rate in this experiment. It may be seen that α is twice as large for the controls than the children with dyslexia for manual responses (0.141 vs 0.073) and one third larger for the foot responses (0.116 vs 0.086). This is a huge difference. Bearing in mind that the learning varies as a function of the time to the power α, if a skill takes a normal child 100 hours to master, it would, taking an average ratio of the learning rates as say 1.5, take a child with dyslexia $100^{1.5}$ i.e. 1000 hours (10 times as long) to learn the skill to the same criterion. Note that the longer the time taken for a normal child to acquire a skill, the greater the predicted decrement - for a skill taking a normal child say 400 hours, it would take a child with dyslexia 20 times as long, and so on.

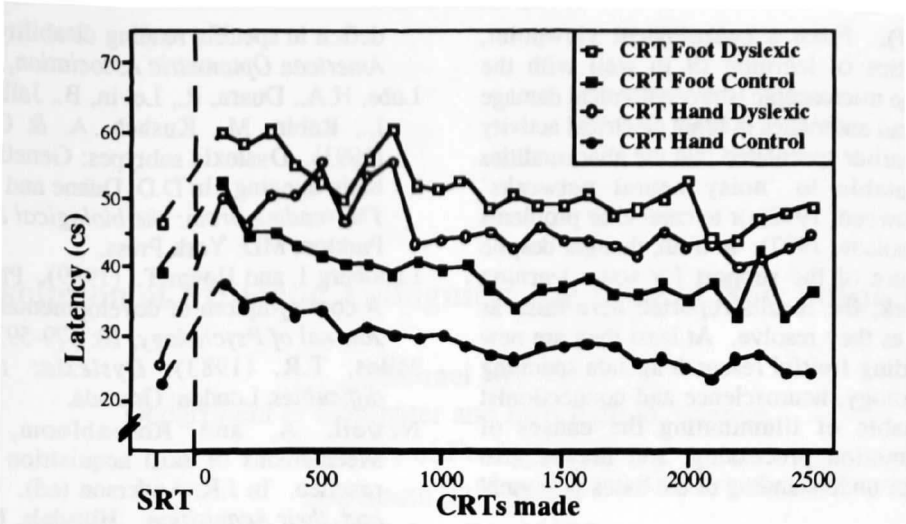

Figure 2. Median Latencies over the period of CRT training

Overall Discussion

The availability of data from training on two quite different skills strengthens the possible interpretation of the results. In particular, it seems reasonable to ascribe the initial performance decrement found in both experiments to a real difficulty in the initial proceduralisation of skill. Furthermore lower 'quality' of automatisation (indexed by speed and by errors) was found in both studies. It seems reasonable, therefore, to argue that this group of children with dyslexia have difficulties with the initial proceduralisation of skill, and with the 'quality' of skill post-training, but that the 'strength' of skill automatisation (as assessed by resistance to unlearning, by ease of relearning, and by dual task performance) is normal. The rate of improvement with practice (α) appeared normal in the Pacman study but was markedly impaired in the CRT training study. This study does not address the interesting issue of how the learning performance of children with dyslexia compares with that of younger normal children of equivalent reading age. We would speculate that, for normal children, although baseline speed increases with age over the period say 8 to 20 years, the learning rate is relatively invariant. If the CRT training results apply to children with dyslexia generally, and apply to tasks other than choice reactions, we are led to a radically new prediction for dyslexic performance, namely that rather than being at the level of children their own age, or even, as is often considered the appropriate control group, children of the same reading age, the performance of children with dyslexia on any task will (in the absence of opportunity to exploit their more mature metaskills, strategies and knowledge) be comparable with that of much younger children, with the amount of impairment increasing as the square root of the necessary learning time.

The framework also has interesting implications for remediation. It would appear from the training studies that children with dyslexia *can* automatise skills and that, once a skill has been automatised, it shows the highly desirable qualities which characterise normal automatic performance, namely greater speed, reduction in effort, resistance to unlearning, and long-term retention, although there may be greater intrinsic variability in the performance. However, slower and more error-prone initial performance militates against the development of automaticity in children with dyslexia. Additionally, if inappropriate methods are acquired, the high resistance of automatised skills to unlearning will make it particularly difficult for children with dyslexia to recover from these early bad habits and learn to perform the skill efficiently. Furthermore, given the reduced learning rate, it is crucial to ensure that the learning is on the right lines. Consequently, it would appear appropriate to concentrate resources on early diagnosis and support for children with dyslexia, with the support concentrating particularly on ensuring consistency of exposition together with rapid and appropriate feedback in order to foster skill automatisation. In other words, it seems likely that traditional teaching methods can prove effective for children with dyslexia, but they need to be applied more carefully and systematically. This conclusion is of course consistent with much recent research (e.g., Bradley and Bryant, 1983; Lundberg and Høien, 1989; Olson, Wise and Rack, 1989) which has demonstrated lasting benefits of early intervention.

It remains to consider the theoretical interpretation of the results. The learning framework leads naturally to connectionist modelling techniques, and indeed connectionist models of spelling errors are now appearing (Brown, 1993). The framework also integrates well with hybrid cognitive architectures such as ACT* (Anderson, 1983) or CAP2 (Schneider and

Detweiler, 1987). From a neurological viewpoint, general difficulties of learning fit in well with the findings of diffuse microscopic neuroanatomical damage and the widespread anomalies in brain electrical activity data. We have earlier speculated that the abnormalities may be attributable to 'noisy neural networks' (Nicolson and Fawcett, 1992) or to cerebellar problems (Fawcett and Nicolson, 1992). In truth, though, despite the striking nature of the support for some learning deficit framework, the results reported here raise as many questions as they resolve. At least they are new questions, providing fruitful research agenda spanning cognitive psychology, neuroscience and connectionist modelling, capable of illuminating the causes of abnormal information processing, and maybe also facilitating deeper understanding of the bases of normal cognition.

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