Available online at www.sciencedirect.com
ScienceDirect
Brain and Language xxx (2008) xxx–xxx
Brain and Language
www.elsevier.com/locate/b&l

Hemispheric dissociation and dyslexia in a computational model of reading ☆

Padraic Monaghan [a,*], Richard Shillcock [b]

[a] Department of Psychology, University of York, York YO10 5DD, UK
[b] Department of Psychology, University of Edinburgh, 7 George Square, Edinburgh EH8 9JZ, UK

Accepted 12 December 2007

Abstract

There are several causal explanations for dyslexia, drawing on distinctions between dyslexics and control groups at genetic, biological, or cognitive levels of description. However, few theories explicitly bridge these different levels of description. In this paper, we review a long-standing theory that some dyslexics' reading impairments are due to impairments in hemispheric transfer. We test this theory in a computational model of reading, implementing anatomical features of the visual system. We demonstrate that, when callosal transfer is impaired, the model reads nonwords as well as an unimpaired model, but reads exception words poorly: a pattern of behaviour similar to surface dyslexia. This computational modelling provides a causal link between brain-based theories of dyslexia to cognitive-level theories that refer specifically to phonological impairments within the reading system.

Keywords: Hemispheric processing; Dyslexia; Computational modelling; Levels of description

1. Introduction

Approximately 5–10% of the population show reading impairment greater than would be predicted by performance on other cognitive tasks (Pennington, 2002). Accounts of these reading impairments can be made at a number of different levels of description (Jackson & Coltheart, 2001), consequently, alternative theories of dyslexia may be describing the same impairment with a consistent aetiology, though the link between levels for describing dyslexia is not yet well understood (for a review see Bishop & Snowling, 2004).

It is generally accepted that a large proportion of developmental dyslexics have phonological impairments (Bishop & Snowling, 2004; Wagner & Torgesen, 1987). In terms of brain functioning, Shaywitz et al. (2003) claim that the phonological deficit is due to a dysfunctional left hemisphere (LH) cortical region involved in phonological processing. Other theories describe the phonological impairment in terms of dysfunction within brain systems that are not language specific, such as the cerebellum (Nicolson, Fawcett, & Dean, 2001), or the visual magnocellular pathways (Lovegrove, Martin, Blackwood, & Badcock, 1980; Stein & Walsh, 1997). An auditory magnocellular deficit, which results in impairment to speech processing, has also been proposed (Bishop, 2007; Tallal et al., 1996).

Evidence for these brain theories is generally derived from correlating dyslexics' reading performance with deficits on other cognitive tasks that depend on the same impaired brain system (Eden & Zeffiro, 1998; Ramus et al., 2003; Vellutino, Fletcher, Snowling, & Scanlon, 2004). However, co-occurrence of deficits does not prove a common cause, and so it is therefore necessary to demonstrate precisely how a brain deficit may impact on cognitive

☆ This research was partially supported by Wellcome Trust Grant 059080. Thanks to Charles Hulme and Maggie Snowling for helpful discussions on this paper.
* Corresponding author. Present address: Department of Psychology, Lancaster University, Lancaster LA1 4YF, UK. Fax: +44 1524 593744.
E-mail address: p.monaghan@lancaster.ac.uk (P. Monaghan).

0093-934X/$ - see front matter © 2007 Elsevier Inc. All rights reserved.
doi:10.1016/j.bandl.2007.12.005

processing in order to establish a direct, causal link between brain-level theories and cognitive impairments.

In this paper we test a direct link between a brain-level theory of dyslexia and its cognitive consequences, namely, that reading impairments are caused by dysfunctional coordination and transfer of information between the two cerebral hemispheres (Geschwind & Galaburda, 1986; Orton, 1925). The hemispheric dissociation theory of dyslexia has a long tradition, drawing on anatomical differences in the brains of dyslexics and controls, and behavioural evidence of similarities in cognitive deficits in dyslexics and individuals with an impaired corpus callosum. We present a computational model that implements this theory, providing an explicit test of how far a direct link can be made between a proposed brain cause of dyslexia and a manifestation of reading deficits at the cognitive level of description.

2. Hemispheric dissociation in dyslexics

Anatomical studies of dyslexic and control brains have suggested reduced cerebral asymmetry in dyslexics (Galaburda, Menard, & Rosen, 1994; Haslam, Dalby, Johns, & Rademaker, 1981; Hier, Le May, Rosenberger, & Perlo, 1978; Rumsey et al., 1986), consistent with Orton's (1925) view that reading difficulties resulted from an impairment in the LHs dominance over the right hemisphere (RH) (Annett, 1996). Dyslexics tend to have a larger RH planum temporale than controls (Beaton, 1997), an area related to language lateralisation (Foundas, Leonard, Gilmore, Fennell, & Heilman, 1994), and close to a region that shows an absence of activity in dyslexics' reading neural network (Temple et al., 2003). Anatomical studies of the corpus callosum have indicated that dyslexics tend to have a larger isthmus and splenium (Duara et al., 1991; Robichon & Habib, 1998), and a smaller posterior midbody and genu than controls (Fine, Semrud-Clikeman, Keith, Stapleton, & Hynd, 2007; Von Plessen et al., 2002), differences that have been related to deficient lateralisation in posterior language-related areas (Rumsey et al., 1999).

There is substantial behavioural evidence that dyslexics have impaired hemispheric transfer. Callosal agenesis is a developmental disorder in which the corpus callosum fails to develop normally, with callosal fibres failing to connect across the hemispheres. Children with callosal agenesis have problems in phonological processing tasks that dyslexics typically find difficult (Temple & Ilsey, 1993; Temple, Jeeves, & Vilarroya, 1990), and both groups have coordination problems in responding with left and right hand, tactile finger localization, pointing to sound sources, and discriminating visually presented lines. Dyslexics and partial or complete commissurotomy patients are also similar in terms of coordinating responses with two hands, disengaging attention from spatial cues, and ERP responses to valid and invalid visual cues (see Mather, 2001, for a review). Tasks that directly test the quality and speed of hemispheric transfer also show differences between dyslexics and controls (e.g.,. Beaumont, Thomson, & Rugg, 1981; Henderson, Barca, & Ellis, 2007).

3. Modelling causes of dyslexia

The distinction between phonological and surface dyslexia subtypes has been most influential in computational models of dyslexia (Bailey, Manis, Pedersen, & Seidenberg, 2004). Children with phonological dyslexia have difficulty in reading nonwords, or novel words, though reading of known words is good. In contrast, surface dyslexic children can read nonwords but tend to over-generalise in the pronunciation of exception words, such as *pint*, *bomb*, or *bind* (Castles & Coltheart, 1993; Stanovich, Siegel, & Gottardo, 1997). Surface dyslexia may be more accurately seen as a reading delay, in that reading of exception words is poor in younger children, but develops over time (Manis, Seidenberg, Doi, McBride-Chang, & Peterson, 1996).

Several computational models of reading have highlighted impairments that may lead to dyslexia. The Dual Route Cascading (DRC) model (Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001) was constructed to embody within its architecture the dissociation between impairments to nonword and exception word reading, by including a route that read each stimulus type. In contrast, researchers in the connectionist tradition have proposed that dyslexia subtypes can result from impairments to a single physical system mapping written forms onto spoken forms without postulating separate routes *a priori* (Seidenberg & McClelland, 1989). In this class of models, phonological dyslexia behaviour results from impairments in the creation of stable phonological representations, and surface dyslexia results from shortage of resources or slowed learning of the mapping of words onto their spoken forms (Harm & Seidenberg, 1999; Plaut, McClelland, Seidenberg, & Patterson, 1996; Seidenberg & McClelland, 1989) However, nonword reading and exception word reading were not entirely dissociable in these models, perhaps reflecting the prevalence of mixed cases, but cases of pure surface dyslexia are beyond their remit (Castles & Coltheart, 1996).

These accounts of surface dyslexia are based on a general quantitative resource limitation; as such they represent a strong claim about cortical plasticity, where the brain is assumed to be unable to solve a mapping problem that is within the abilities of computational models containing only 100 interconnected units. Although such accounts successfully demonstrate the relative vulnerability of exception words, they are underspecified with respect to any neuropsychological basis for the resource limitation. The same argument may be applied to "division of labour" accounts of dissociations in dyslexia, where surface dyslexia is due to greater reliance on reading via semantics rather than directly from orthography to phonology. Although the more arbitrary, idiosyncratic relationship between the respective orthographic and phonological representations of "yacht" or "pint" may tilt the processor away from

Please cite this article in press as: Monaghan, P., & Shillcock, R., Hemispheric dissociation and dyslexia in a computational ..., *Brain and Language* (2008), doi:10.1016/j.bandl.2007.12.005

complete reliance on the phonological route compared with words with regular pronunciations, this mapping is again a relatively trivial computational problem compared with the massive arbitrariness that the brain copes with in the rest of the lexicon, for instance in mapping words' phonology onto semantics.

Our model of dyslexia builds on the connectionist modelling tradition, but we instantiate anatomical features of the visual system in the model in assuming the minimal architecture necessary given by the observable anatomy of the brain. The most compelling account of reading phenomena is one in which the required behaviour emerges from the structure of the problem that is already given in the interaction between the information structure of the lexicon and the architecture of the cognitive system. The model is illustrated in Fig. 1. The critical innovation is instantiating two sets of hidden units, representing resources in the LH and RH, with connections between these sets of units representing the corpus callosum.

In the brain, visual input from the left visual field (LVF) projects initially to the RH, and input from the right visual field (RVF) projects initially to the LH. This contralateral projection is precisely defined in that the fovea, the high-resolution centre of the visual field, also demonstrates this divided projection to the left and right cortices (see, *e.g.*,

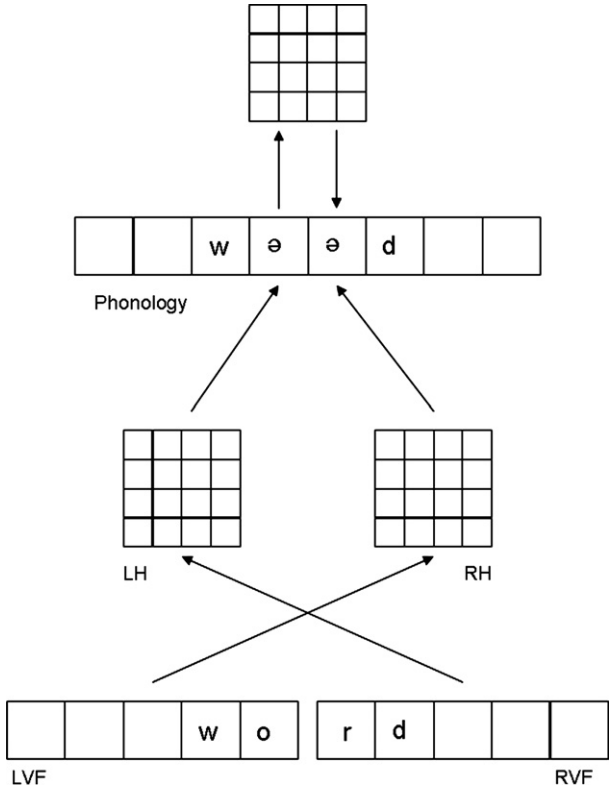

Fig. 1. The hemispheric model of reading, mapping orthographic to phonological forms for monosyllabic words. Words are presented at each position in the input, and the input to the left and right of the model is contralaterally projected to the hidden layers. LVF, left visual field input to the model; RVF, right visual field input; LH, set of units in the left hidden layer; RH, set of units in the right hidden layer.

Fendrich & Gazzaniga, 1989; Lavidor & Walsh, 2004; Leff, 2004). Isolated words are processed with greatest facility when fixated slightly to the left of centre, consequently, the visual information about the word is initially divided equitably between the LH and RH (Brysbaert, 1994; Shillcock, Ellison, & Monaghan, 2000).

A critical question is what effect this initial division of the visual field has on visual word processing. At some point the orthographic information in the two hemifields has to be combined to specify the identity of the word and to access its phonological form and its meaning. The point at which this integration occurs is a matter of debate, yet the psycholinguistic and attentional effects of this division are seen in higher levels of language processing indicating that the division has a profound influence (Ellis, Brooks, & Lavidor, 2005; Hsiao & Shillcock, 2005; Jung-Beeman, 2005; Lavidor & Ellis, 2002, 2003; Lavidor, Hayes, Shillcock, & Ellis, 2004; Young & Ellis, 1985). Elsewhere, we have explored the implications of this initial splitting for visual word recognition in theoretical (Shillcock et al., 2000), and connectionist (Shillcock & Monaghan, 2001a) models of normal reading, and such effects have also been successfully simulated in the SERIOL model of reading with initial division of visual processing between the two hemispheres (Whitney & Cornelissen, 2004).

Reading exception words in particular requires integration of the orthographic information in each hemifield to occur prior to pronunciation. If the orthographic information is not effectively integrated before the point at which the phonological representation is formed then the phonological representation will be formed componentially and regularisation errors will tend to occur. Consider the word *pint*, fixated close to the optimal viewing position as *pi*nt*, where * indicates the fixation point. (For convenience, we do not consider the splitting of individual letters in fixation; such splitting does not materially affect the case we make.) There are 14 English word lemmas of length 4 beginning with *pi* (pied, pimp, ping, pith, piss, pike, pier, pint, pine, pill, pipe, pink, pile, and pick). Of these, 7 have a vowel pronunciation /I/, 6 /aI/, and 1 /Iə/. Attempting to predict the vowel from the last letters (nt) is harder still: there are 25 word lemmas ending nt, and 8 different vowel pronunciations are possible: /æ/, /ɑ:/, /aI/, /əʊ/, /ɛ/, /I/, /ɒ/, and /Λ/. Hence, information about the onset and the coda has to be combined before the vowel can be pronounced. If transfer is impaired, then the most frequent vowel consistent with the *pi* and *nt* pairing is most likely to be accessed, which would be /I/, a classic instance of over-generalisation.

Formally, reading exception words is akin to the XOR problem, a class of linearly inseparable problems. A connectionist model with split input and with no interaction between the two halves before the output is equivalent to a perceptron and will not be able to solve the mapping (Minsky & Papert, 1969), and exception word reading will be particularly impaired without adequate hemispheric transfer. However, as is evident from Manis et al's.

Please cite this article in press as: Monaghan, P., & Shillcock, R., Hemispheric dissociation and dyslexia in a computational ..., *Brain and Language* (2008), doi:10.1016/j.bandl.2007.12.005

(1996) study, most children with surface dyslexia are poorer in reading regular words, though nonword reading is relatively intact. With hemispheric dissociation of visual information, if there is inadequate combination of letter information from the LVF and RVF then, even for regular words, reading will be somewhat impaired. In the case of the regular word *mint*, for example, fixated as *mi*nt*, pronunciations of words consistent with *mi* will be partially activated in one half of the system, and words consistent with *nt* will be activated in the other half. The word *pint* is among this set, and so the vowel /aI/ will be partially activated. Thus, hemispheric dissociation provides a potential account of surface dyslexia, and predicts that it generally involves some small deficit in reading regular words.

In the first set of models, we adapted Harm and Seidenberg's (1999) connectionist model of reading by incorporating a divided visual field and two banks of units representing the two hemispheres. We also tested an impaired version of this model, by impeding the transfer of information between its two halves. The third simulation tested the effect of increasing resources on normal and impaired reading performance in the model.

4. Simulations 1 and 2: Normal and impaired reading with divided visual input

The model employed in this set of simulations was a variation on the Harm and Seidenberg (1999) model, with two innovations: (1) orthographic input to the model was presented according to information about fixation positions of words during reading[1]; and (2) the visual field was divided into a left and right half, and projected contralaterally to a divided hidden layer.

4.1. Architecture

The input layer of the model was divided into two sets of units, representing the LVF and RVF (Fig. 1). Each half of the input layer had five letter slots, each comprising 26 units, to represent individual letters of the input. In Fig. 1, *word* is fixated between the 2nd and 3rd letters. The hidden layer was also divided into two sets of 100 U, representing the LH and RH. The left input units were fully connected to the right hidden units, and the right input was fully connected to the left hidden units to reflect the initial projection of the visual fields onto the contralateral hemispheres. In Fig. 1, arrows between layers indicate connectivity. The hidden units were fully connected to an output layer, where the phonological form of the input word was represented. The output layer was composed of 8 phoneme slots: 3 slots for the onset, 2 for the nucleus and 3 for the coda of monosyllabic words. Each slot contained 11 units, representing phonological features as used by Harm and Seidenberg (1999), with values between −1 and 1. Empty slots were represented by 11 features with a −1 target activation. We extended Harm and Seidenberg's phonological representations to British English by including more British-English diphthongs, adding /ə/, and using more slots for onset and coda. Each output unit was self-connected with a weight set at .75, resulting in decay of activation over time. The output layer was connected to a set of 25 units that acted as clean-up units for the phonological representation at the output and also countered the decay in activation resulting from the output layer's self-connections.

In the normal model of reading (Simulation 1) we fully connected the two sets of hidden units to each other. In the model with impaired callosal connectivity (Simulation 2) we omitted these connections but included self-connections between units within each hidden layer, such that each hidden unit was connected to all other hidden units in the same layer. These patterns ensured that the number of connections in each model was the same, and also allowed the same degree of recurrence to occur in each model.

4.2. Training and testing

The model learned to map orthography onto phonology for all 3573 monosyllabic wordforms of length five or less with frequency greater than one per million in the CELEX database (Baayen, Pipenbrock, & Gulikers, 1995). Words were presented in random order, according to their log-compressed frequency (Plaut et al., 1996), and occurred in all possible fixation positions, as indicated in Fig. 2, resulting in 18,910 training patterns. In naturalistic reading, words are fixated at any and all positions during reading of text with a slight preference for fixations towards word centre, though we do not implement this fixation preference as it is very slight for short words (Brysbaert &

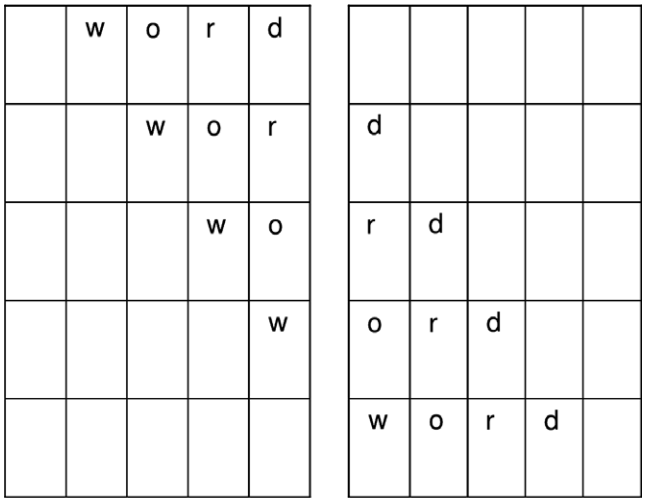

Fig. 2. The complete range of word inputs for four letter words to the hemispheric reading model.

[1] We also tested a version of the model with words at the input presented at a single position, aligned at the vowel, as in Harm and Seidenberg's (1999) model. We found that this training regime resulted in similar behaviour to that of the models presented here, both for normal and impaired versions of the model (Shillcock & Monaghan, 2001b).

Vitu, 1998). We make the simplifying assumption that words are fixated an equal number of times in each position, and so presentation position was randomly selected.

We pre-trained the model's phonological attractors in a "listening task" by presenting the phonological form for words at the output, and allowing the model 5 time intervals to reproduce the phonology. The learning rate was .001, and 1 million listening trials were presented to the model, after which the model's performance was 100% accurate. We then trained the model to map orthography to phonology using recurrent backpropagation over 7 time intervals. At time 1, the input representation of the word was introduced. At time 2, activation passed from input to hidden units. At time 3, the target output representation for the word was presented, and activation passed from the hidden units to the output units, and between the hidden units. For time steps 4 to 7, activation passed between the output layer and the phonological attractors layer, and continued to pass between the hidden units and from the hidden units to the output layer. The learning rate was .005, and 10 million word tokens were presented.

The model was tested by assessing the phonological feature representation of the word at the output at each phoneme slot position. The Euclidean distance from the model's actual output to all possible phonemes and the empty slot representation was computed. The model's production was taken to be the phoneme corresponding to the smallest distance for each slot. The model was judged to have read the word correctly if all 8 slots corresponded to their target phoneme.

To test nonword reading, we took the 357 nonwords of length 5 letters or less used by Harm and Seidenberg (1999). We also tested the model on the 48 exception words from Taraban and McClelland (1987). For all tests, accuracy was tested at all presentation positions for each word. We repeated each simulation 8 times.

4.3. Results and discussion

Fig. 3a shows performance in Simulation 1—the connected hemispheres model—on reading words, nonwords and exception words. After 10 million patterns the model performed well, with 95.5% of the words read correctly. Exception words were 86.0% correct. The connected hemispheres model read 73.1% of the nonwords appropriately, comparable to the 79% in Harm and Seidenberg's (1999) simulations. The hemispheric model's performance was slightly lower than that of Harm and Seidenberg's (1999) for overall reading accuracy and nonword reading; this difference was due principally to the larger training set (18,910 patterns compared with 3123) and the more complex phonological representation (8 phoneme slots compared with 6), rather than to the different architecture used. A hemispheric model trained on a word set comparable to that of Harm and Seidenberg's model learned to 99% correct after five million presentations (Shillcock & Monaghan, 2001b). The connected model of reading therefore learns

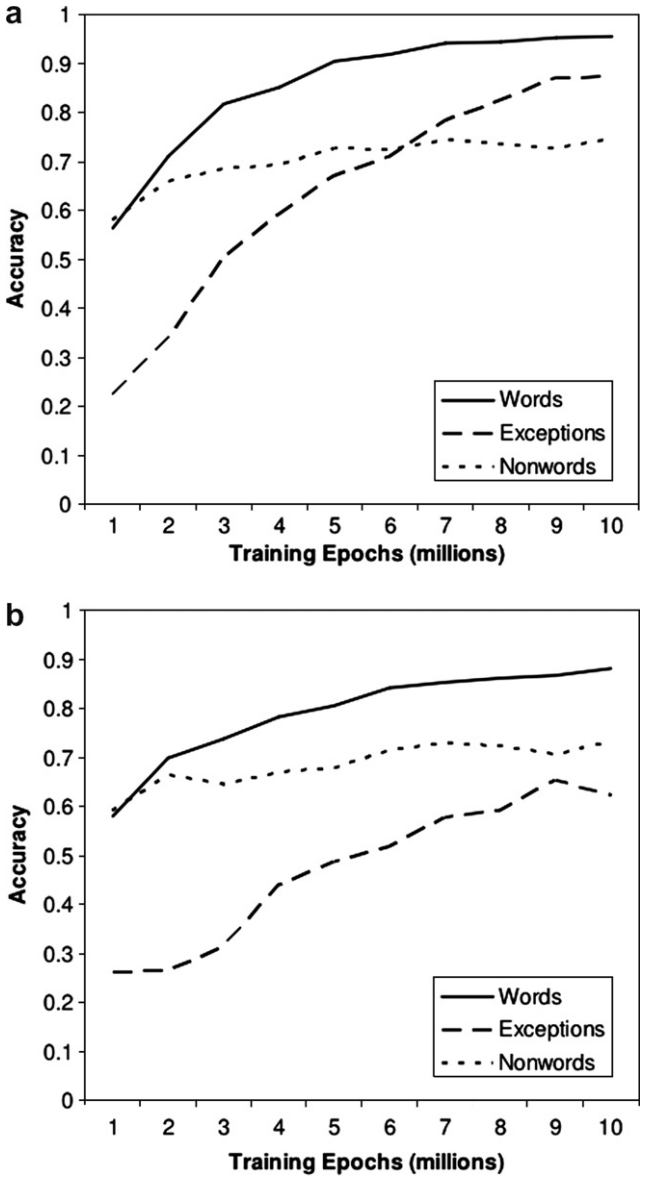

Fig. 3. The performance of the hemispheric reading model with phonological attractors on reading all words in the training set, reading exception words, and generalisation to nonword reading. (a) The normal model's performance, with hemispheric connections intact. (b) The impaired model's performance, with no inter-hemispheric connections.

to read the training set accurately, and demonstrates generalisation to new words similar to that of other connectionist models of reading, providing a good basis for testing impairments to the structure of the model.

Fig. 3b shows the results of Simulation 2, the disconnected hemispheres model. As predicted, the model's performance is worse than the connected model on reading words, with 87.8% of the training set read correctly, $t(14) = 18.49$, $p < .001$. The reading of nonwords was at a level comparable to that of the connected model, with 71.8% read correctly, though marginally significantly less accurate, $t(14) = 1.81$, $p = .09$. Also as predicted, the disconnected model read exception words substantially less

accurately than the connected model, reading 62.4% correctly, $t(14) = 32.33$, $p < .001$. Table 1 provides examples of exception word reading by the disconnected model at the end of training. The errors are typically over-regularisations, of a type characteristic of surface dyslexics (e.g., Manis et al., 1996).

The consequences of impaired transfer between the two sets of hidden units are therefore multiple. Performance overall is slightly poorer on the whole set of words used for training compared with the connected model, yet performance on nonwords was not substantially affected by this disconnection, indicating that generalization relied on "componential" processing. Yet, the most striking effect was on the exception words, where performance was much lower in the disconnected version of the model. This result is achieved without introducing parametric constraints, such as a reduced learning rate or reduced hidden layer resources. The impairment to reading exception words is due to the architecture of the disconnected model.

The model learned to read each word in the training set in all possible presentation positions. Yet, as with the SERIOL model of reading (Whitney & Cornelissen, 2004), the similar properties of the same word presented in different positions was discovered by the model. Hence, if the model made a reading error in one position, it was likely to make the same error in other presentation positions: errors at only one presentation position accounted for 20.1% and 11.8% of the connected- and disconnected-hemispheres models' errors, respectively. Consistent with the additional processing load of single hemisphere processing of stimuli (Monaghan & Pollmann, 2003; Weissman & Banich, 2000), there was no evidence for a systematic advantage for reading words unilaterally in the model. For the connected hemispheric model for all words of length greater than 2, 95.0% of words were read correctly in the LVF and in the RVF, and 95.7% were read correctly when presented across the visual fields, which was significantly greater than RVF presentations, $t(3569) = 3.35$, $p < .001$, but not greater than LVF presentations, $t(3569) = 1.03$, $p = .30$. In the disconnected model, 78.0% presented to the LVF were read correctly, 77.0% presented to the RVF were read correctly, and 77.1% of medially presented words were read correctly. None of the proportions correct differed significantly by presentation position, all $t(3569) < 1$. We predict that there would be little change in performance for surface dyslexic children for lateralised word reading, though we know of no studies that have tested this.

Several connectionist models have reproduced the surface dyslexia subtype by reducing the resources available for the mapping from orthographic representations to phonological representations (e.g., Harm & Seidenberg, 1999; Seidenberg & McClelland, 1989). We tested whether the behaviour of our models was due to resource limitations in the model without "callosal" connections by increasing the number of units in the hidden layer, or whether the anatomical distinction in the model alone accounted for the effect.

Table 1
Performance of the impaired hemispheric model on exception word reading

Word	Target pronunciation	Model's pronunciation
come	/kʊm/	/kʊʊm/
foot	/fʌt/	/fuːt/
shoe	/ʃuː/	/ʃəu/
pint	/paɪnt/	/pɪɪnt/
deaf	/dɛf/	/diːf/
pear	/pɛəɹ/	/pɪəɹ/
shall	/ʃæl/	/ʃɔːl/
have	/hæv/	/hɛɪv/
does	/dʌz/	/dəuz/

5. Simulation 3: Effect of increasing resources

5.1. Architecture

Simulation 3 was identical to Simulation 2, the disconnected hemispheres model, except that we increased the hidden layer resources from 100 per side to 200.

5.2. Training and testing

The model was trained and tested in the same way as in simulations 1 and 2.

5.3. Results and discussion

Fig. 4 shows the model's performance on all words, exception words, and nonwords. After 10 million patterns, performance on the whole set of words was 92.3%, and

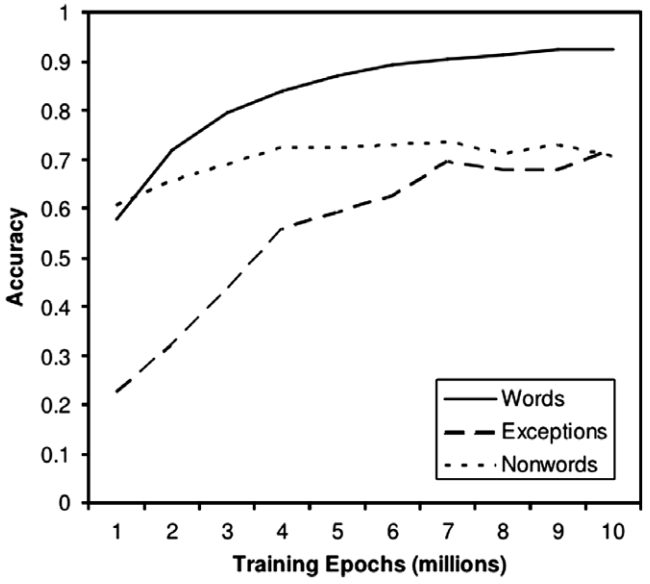

Fig. 4. Performance of the impaired hemispheric reading model with 200 hidden units on reading all words, exception words, and nonwords.

Please cite this article in press as: Monaghan, P., & Shillcock, R., Hemispheric dissociation and dyslexia in a computational ..., *Brain and Language* (2008), doi:10.1016/j.bandl.2007.12.005

nonword performance remained at a similar level as in the disconnected model with 100 hidden units, with 70.7% read appropriately. As predicted, exception word reading remained much poorer than reading of the whole word set, at 72.2% correct. Though increasing resources resulted in greater accuracy compared with the 62.2% correct for the disconnected model with 100 hidden units, it was still substantially lower than the 87.4% correct for the connected model. Hence, increasing resources did not qualitatively alter the impaired hemispheric model's performance. Taken with the results of Simulations 1 and 2, the architectural principle underlying the surface dyslexia behaviour is relatively impervious to parametric variations in resources for forming the mapping. The nature of the linearly inseparable problem of reading exception words is unaffected by more resources, as the difficulty is due primarily to information not being integrated before the output stage.

6. General discussion

The range of studies demonstrating behavioural similarities between dyslexics and patients with callosal impairment, and the anatomical studies demonstrating morphological aberrations in the corpus callosum of dyslexics, suggest that dyslexia and hemispheric dissociation are correlated. The computational modelling we have described provides an explicit link between different levels of description and shows how a brain level impairment may cause a cognitive level impairment, going beyond simply demonstrating a correlation. The computational models instantiated the informational constraints on reading resulting from such impaired callosal transfer, which we have demonstrated to be sufficient to cause surface dyslexia.

The quantitative results of the modelling have shown that impairments in transfer of information between the hemispheres during the mapping of orthography onto phonology is sufficient to affect to some degree performance on all words in the training set, to delay or prevent learning of exception words in particular, without also entailing a substantial reduction in accuracy of reading nonwords. This impairment does not happen within the phonological representations, but is due to problems in forming the mapping between written and spoken forms of words. Deficits within the phonological system itself may contribute additionally to dyslexic behaviour. Indeed, we have not attempted to provide a model of phonological dyslexia. Within the same framework we have used for our modelling, impairments to the phonological attractors within the model have been shown to result in specific impairment to reading nonwords (Harm & Seidenberg, 1999) and our own model should be similarly extended.

We have, however, provided a detailed account of surface dyslexia, which, in contrast to other connectionist models of surface dyslexia, is not a resource-based account. Instead, the exception word reading impairment results from integration of information between the two hemispheres of the model at a stage too late in the pathway from orthography to phonology for effective integration of information. We have thus provided an upper-bound on the point at which information about the word must interact within the reading system. If this interaction is inadequate or inefficient, then reading of exception words will be affected in particular, and general reading performance will be somewhat reduced. Critically, such an account is not due to parametric differences in the model. Increasing resources in the left and right hidden layer of the model with no ''callosal'' connections has no substantial impact on the pace of learning to read in the model, as indicated in Simulation 3.

We created the hemispheric model within the connectionist tradition, adapting Harm and Seidenberg's (1999) model in order to inherit the broad range of reading phenomena which it produces. Our account differs in that we show how anatomical pathways relevant to the reading system can provide additional constraints to capture reading impairments, and can—unlike Harm and Seidenberg's (1999) model—generate pure cases of surface dyslexia. However, other traditions of modelling reading are also compatible with the principles we have presented in our hemispheric model, and we make no special case for the particular architecture we have used, but rather highlight the value of incorporating anatomical information as input to reading models. The DRC model of reading (Coltheart et al., 2001; Perry, Ziegler, & Zorzi, 2007) simulated surface dyslexia by impairing processing of the lexical route, and it is perfectly possible that the *input* to this system would be affected more substantially by impairments to unifying the left and right visual information about the word, as compared with impairments to a grapheme-phoneme correspondence system, where all information about the word does not have to be available simultaneously in order to pronounce the word correctly. Our modelling, therefore, provides insight into the influence of early stages of visual processing on the reading system, in particular, the requirement to integrate initially divided visual information.

A consequence of inadequate transfer of information sufficiently early in the orthography to phonology mapping is that the RH of the model is implicated in phonological processing with some independence from the LH (Galaburda et al., 1994). Consequently, the modelling predicts reduced activation in LH phonological processing and increased activation in the corresponding regions of the RH in dyslexics. This prediction is supported by imaging studies of reduced activity in the LH temporo-parietal and increased activity in the RH cortex of dyslexics (Brunswick, McCrory, Price, Fritch, & Frith, 1999; Horwitz, Rumsey, & Donohue, 1998; Paulesu et al., 1996; Pugh et al., 2001; Rumsey et al., 1999; Shaywitz, Lyon, & Shaywitz, 2006; Temple et al., 2003).

The model of surface dyslexia based on disconnected hemispheres does not necessarily predict that dyslexics will experience no reading problems if words are projected exclusively to one hemifield, such as the RVF. Normal word reading overwhelmingly involves parafoveal preview

Please cite this article in press as: Monaghan, P., & Shillcock, R., Hemispheric dissociation and dyslexia in a computational ..., *Brain and Language* (2008), doi:10.1016/j.bandl.2007.12.005

followed by fixation within the word, implicating hemispheric storage and transfer and necessitating the appropriate partial representations within each hemisphere, though the model's generalisation across different presentation positions for the same word indicates that divided processing is a feature of lexical processing even when words are unilaterally presented (see Pollmann, Zaidel, & von Cramon, 2004, for fMRI evidence of a similar effect in simple letter processing tasks). The brain typically operates best when able to bring the resources of both hemispheres to bear on a problem (Weissman & Banich, 2000), and we have argued elsewhere (Shillcock et al., 2000) that aspects of normal reading behaviour can be understood in terms of ensuring an equitable division of labour between the two hemispheres. The limited data available—from the mean initial landing position on four-letter words in sentences (Kelly, Jones, McDonald, & Shillcock, 2004)—suggest that some dyslexics may adopt reading behaviours that shift their fixation of a word slightly leftwards, thereby projecting more of the word to the LH, and reducing the need for hemispheric transmission of information. This behaviour was not a side-effect of more fixations for dyslexics, as no differences in the size of saccadic movements forward through the text were found between control and dyslexic readers.

It is one of the goals of cognitive neuroscientists to ground cognitive phenomena in the observable anatomical substrate of the brain. The division of the brain into two hemispheres is the largest anatomical distinction within the brain. We have made a first step towards the goal by showing that this fundamental architectural distinction can be implemented in computational models of reading, thereby effectively linking the brain-based theory of dyslexia as hemispheric dissociation to disruption within the cognitive representations involved in reading.

References

Annett, M. (1996). In defence of the right shift theory. *Perceptual and Motor Skills, 82*, 115–137.

Baayen, R. H., Pipenbrock, R., & Gulikers, L. (1995). *The CELEX lexical database. Linguistic Data Consortium*. Philadelphia, PA: University of Pennsylvania.

Bailey, C. E., Manis, F. R., Pedersen, W. C., & Seidenberg, M. S. (2004). Variation among developmental dyslexics: Evidence from a printed-word-learning task. *Journal of Experimental Child Psychology, 87*, 125–154.

Beaton, A. A. (1997). The relation of planum temporale asymmetry and morphology of the corpus callosum to handedness, gender, and dyslexia: A review of the evidence. *Brain and Language, 60*, 255–322.

Beaumont, J. G., Thomson, M., & Rugg, M. D. (1981). An intrahemispheric integration deficit in dyslexia. *Current Psychological Research, 1*, 185–198.

Bishop, D. V. M. (2007). Using mismatch negativity to study central auditory processing in developmental language and literacy impairments: where are we, and where should we be going? *Psychological Bulletin, 133*, 651–672.

Bishop, D. V. M., & Snowling, M. J. (2004). Developmental dyslexia and specific language impairment: Same or different? *Psychological Bulletin, 130*, 858–886.

Brunswick, N., McCrory, E., Price, C. J., Fritch, C. D., & Frith, U. (1999). Explicit and implicit processing of words and pseudowords by adult developmental dyslexics: A search for Wernicke's Wortschatz. *Brain, 122*, 1901–1917.

Brysbaert, M. (1994). Interhemispheric transfer and the processing of foveally presented stimuli. *Behavioural Brain Research, 64*, 151–161.

Brysbaert, M., & Vitu, F. (1998). Word skipping: Implications for theories of eye movement control in reading. In G. Underwood (Ed.), *Eye guidance in reading and scene perception*. Amsterdam: Elsevier.

Castles, A., & Coltheart, M. (1993). Varieties of developmental dyslexia. *Cognition, 47*, 149–180.

Castles, A., & Coltheart, M. (1996). Cognitive correlates of developmental surface dyslexia: A single case study. *Cognitive Neuropsychology, 13*, 25–50.

Coltheart, M., Rastle, K., Perry, C., Langdon, R., & Ziegler, J. (2001). DRC: A dual route cascaded model of visual word recognition and reading aloud. *Psychological Review, 108*, 204–256.

Duara, R., Kushch, A., Gross-Glenn, K., Barker, W. W., Jallad, B., Pascal, S., et al. (1991). Neuroanatomic differences between dyslexic and normal readers on magnetic resonance imaging scans. *Archives of Neurology, 48*, 410–416.

Eden, G. F., & Zeffiro, T. A. (1998). Neural systems affected in developmental dyslexia revealed by functional neuroimaging. *Neuron, 21*, 279–282.

Ellis, A. W., Brooks, J., & Lavidor, M. (2005). Evaluating a split fovea model of visual word recognition: Effects of case alternation in the two visual fields and in the left and right halves of words presented at the fovea. *Neuropsychologia, 43*, 1128–1137.

Fendrich, R., & Gazzaniga, M. S. (1989). Evidence of foveal splitting in a commissurotomy patient. *Neuropsychologia, 27*, 273–281.

Fine, J. G., Semrud-Clikeman, M., Keith, T. Z., Stapleton, L. M., & Hynd, G. W. (2007). Reading and the corpus callosum: An MRI family study of volume and area. *Neuropsychology, 21*, 235–241.

Foundas, A. L., Leonard, C. M., Gilmore, R., Fennell, E., & Heilman, K. M. (1994). Planum temporale asymmetry and language dominance. *Neuropsychologia, 32*, 1225–1231.

Galaburda, A., Menard, M., & Rosen, G. (1994). Evidence for aberrant auditory anatomy in developmental dyslexia. *Proceedings of the National Academy of Sciences, 91*, 8010–8013.

Geschwind, N., & Galaburda, A. M. (1986). *Cerebral lateralization: Biological mechanisms associations, and pathology*. Cambridge, MA: MIT Press.

Harm, M. W., & Seidenberg, M. S. (1999). Phonology, reading acquisition, and dyslexia: insights from connectionist modelling. *Psychological Review, 106*, 491–528.

Haslam, R. H. A., Dalby, J. T., Johns, R. D., & Rademaker, A. W. (1981). Cerebral asymmetry in developmental dyslexia. *Archives of Neurology, 38*, 679–682.

Henderson, L., Barca, L., & Ellis, A. W. (2007). Interhemispheric cooperation and non-cooperation during word recognition: Evidence for callosal transfer dysfunction in dyslexic adults. *Brain and Language, 103*, 276–291.

Hier, D. B., Le May, M., Rosenberger, P. M., & Perlo, V. P. (1978). Developmental dyslexia. Evidence for a subgroup with a reversal of cerebral asymmetry. *Archives of Neurology, 35*, 90–92.

Horwitz, B., Rumsey, J. M., & Donohue, B. C. (1998). Functional connectivity of the angular gyrus in normal reading and dyslexia. *Proceedings of the National Academy of Sciences, 95*, 8939–8944.

Hsiao, J. H., & Shillcock, R. (2005). Foveal splitting causes differential processing of Chinese orthography in the male and female brain. *Cognitive Brain Research, 25*, 531–536.

Jackson, N. E., & Coltheart, M. (2001). *Routes to reading success and failure: Toward an integrated cognitive psychology of atypical reading*. Hove, UK: Psychology Press.

Jung-Beeman, M. (2005). Bilateral brain processes for comprehending natural language. *Trends in Cognitive Sciences, 9*, 512–518.

Please cite this article in press as: Monaghan, P., & Shillcock, R., Hemispheric dissociation and dyslexia in a computational ..., *Brain and Language* (2008), doi:10.1016/j.bandl.2007.12.005

Kelly, M. L., Jones, M. W., McDonald, S. A., & Shillcock, R. C. (2004). Dyslexics' eye fixations may accommodate to hemispheric desynchronisation. *NeuroReport, 15*, 2629–2632.

Lavidor, M., & Ellis, A. W. (2002). Word length and orthographic neighborhood size effects in the left and right cerebral hemispheres. *Brain and Language, 80*, 45–62.

Lavidor, M ., & Ellis, A. W. (2003). Interhemispheric integration in a split-processing model of visual word recognition. *Cortex, 39*, 69–83.

Lavidor, M., Hayes, A., Shillcock, R., & Ellis, A. W. (2004). Evaluating a split processing model of visual word recognition: Effects of orthographic neighborhood size. *Brain and Language, 88*, 312–320.

Lavidor, M., & Walsh, V. (2004). The nature of foveal representation. *Nature Reviews Neuroscience, 5*, 729–735.

Leff, A. (2004). A historical review of the representation of the visual field in primary visual cortex with special reference to the neural mechanisms underlying macular sparing. *Brain and Language, 88*, 268–278.

Lovegrove, W., Martin, F., Blackwood, M., & Badcock, D. (1980). Specific reading difficulty: Differences in contrast sensitivity as a function of spatial frequency. *Science, 210*, 439–440.

Manis, F., Seidenberg, M., Doi, L., McBride-Chang, C., & Peterson, A. (1996). On the basis of two subtypes of developmental dyslexia. *Cognition, 58*, 157–195.

Mather, D. S. (2001). Does dyslexia develop from learning the alphabet in the wrong hemisphere? A cognitive neuroscience analysis. *Brain and Language, 76*, 282–316.

Minsky, M. L., & Papert, S. A. (1969). *Perceptrons*. Cambridge, MA: MIT Press.

Monaghan, P., & Pollmann, S. (2003). Division of labour between the hemispheres for complex but not simple tasks: An implemented connectionist model. *Journal of Experimental Psychology: General, 132*, 379–399.

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

Orton, S. T. (1925). "Word-blindness" in school children. *Archives of Neurology and Psychiatry, 14*, 581–615.

Paulesu, E., Frith, U., Snowling, M., Gallagher, A., Morton, J., Frackowiak, R. S., et al. (1996). Is developmental dyslexia a disconnection syndrome? Evidence from PET scanning. *Brain, 119*, 143–157.

Pennington, B. F. (2002). *The development of psychopathology: Nature or nurture*. New York: Guilford Press.

Perry, C., Ziegler, J. C., & Zorzi, M. (2007). Nested incremental modeling in the development of computational theories: The CDP+ model of reading aloud. *Psychological Review, 114*, 273–315.

Plaut, D. C., McClelland, J. L., Seidenberg, M. S., & Patterson, K. (1996). Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychological Review, 103*, 56–115.

Pollmann, S., Zaidel, E., & von Cramon, D. Y. (2004). The neural basis of the bilateral distribution advantage. *Experimental Brain Research, 221*, 322–333.

Pugh, K. R., Mencl, W. E., Jenner, A. R., Katz, L., Frost, S. J., Lee, J. R., et al. (2001). Neurobiological studies of reading and reading disability. *Journal of Communication Disorders, 34*, 479–492.

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

Robichon, F., & Habib, M. (1998). Abnormal callosal morphology in male adult dyslexics: Relationships to handedness and phonological abilities. *Brain and Language, 62*, 127–146.

Rumsey, J. M., Dorwart, R., Vermess, M., Denckla, M. B., Kruesi, M. J., & Rapoport, J. L. (1986). Magnetic resonance imaging of brain anatomy in severe developmental dyslexia. *Archives of Neurology, 43*, 1045–1046.

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.

Seidenberg, M. S., & McClelland, J. L. (1989). A distributed, developmental model of word recognition and naming. *Psychological Review, 96*, 523–568.

Shaywitz, B. A., Lyon, G. R., & Shaywitz, S. E. (2006). The role of functional magnetic resonance imaging in understanding reading and dyslexia. *Developmental Neuropsychology, 30*, 613–632.

Shaywitz, S. E., Shaywitz, B. A., Pugh, K. R., Fulbright, R. K., Skudlarski, O., Mencl, W. E., et al. (2003). Neural systems for compensation and persistence: Young adult outcome of childhood reading disability. *Biological Psychiatry, 54*, 25–33.

Shillcock, R., Ellison, M. T., & Monaghan, P. (2000). Eye-fixation behaviour, lexical storage and visual word recognition in a split processing model. *Psychological Review, 107*, 824–851.

Shillcock, R. C., & Monaghan, P. (2001a). The computational exploration of visual word recognition in a split model. *Neural Computation, 13*, 1171–1198.

Shillcock, R.C. & Monaghan, P. (2001b). Connectionist modelling of surface dyslexia based on foveal splitting: Impaired pronunciation after only two half *pints*. In *Proceedings of the 23rd Annual Conference of the Cognitive Science Society*, pp. 916–921. Mahwah, NJ: Lawrence Erlbaum.

Stanovich, K. E., Siegel, L. S., & Gottardo, A. (1997). Converging evidence for phonological and surface subtypes of reading disability. *Journal of Educational Psychology, 89*, 114–127.

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

Tallal, P., Miller, S. L., Bedi, G., Byma, G., Wang, X., Nagarajan, S. S., et al. (1996). Language comprehension in language-learning impaired children improved with acoustically modified speech. *Science, 27*, 81–83.

Taraban, R., & McClelland, J. L. (1987). Conspiracy effects in word pronunciation. *Journal of Memory and Language, 26*, 608–631.

Temple, C. M., & Ilsey, J. (1993). Phonemic discrimination in callosal agenesis. *Cortex, 29*, 341–348.

Temple, C. M., Jeeves, M. A., & Vilarroya, O. O. (1990). Reading in callosal agenesis. *Brain and Language, 39*, 235–253.

Temple, E., Deutsch, G. K., Poldrack, R. A., Miller, S. L., Merzenich, M. M., & Gabrieli, J. D. E. (2003). Neural deficits in children with dyslexia ameliorated by behavioural remediation: Evidence from functional MRI. *Proceedings of the National Academy of Sciences, 100*, 2860–2865.

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

Von Plessen, K., Lundervold, A., Duta, N., Heiervang, E., Klauschen, F., Smievoll, A. I., et al. (2002). Less developed corpus callosum in dyslexic subjects—a structural MRI study. *Neuropsychologia, 47*, 1035–1044.

Wagner, R. K., & Torgesen, J. K. (1987). The nature of phonological processing and its causal role in the acquisition of reading skills. *Psychological Bulletin, 101*, 192–212.

Weissman, D. H., & Banich, M. T. (2000). The cerebral hemispheres cooperate to perform complex but not simple tasks. *Neuropsychology, 14*, 41–59.

Whitney, C., & Cornelissen, P. L. (2004). Letter-position encoding and dyslexia. *Journal of Research in Reading, 28*, 274–301.

Young, A. W., & Ellis, A. W. (1985). Different methods of lexical access for words presented in the left and right visual hemifields. *Brain and Language, 24*, 326–358.

Please cite this article in press as: Monaghan, P., & Shillcock, R., Hemispheric dissociation and dyslexia in a computational ..., *Brain and Language* (2008), doi:10.1016/j.bandl.2007.12.005